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TREATMENT OF THE TUBERCULOUS PSOAS ABSCESS

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The tuberculous abscess is one of the serious complications of tuberculosis of the spine. It is a local manifestation of a general tuberculous infection, and particularly of osseous involvement. It occurs in about twenty-five per cent of all cases of tuberculous spine infections. The cervical, thoracic and lumbar regions are sites for its occurrence. The abscess may become large enough to cause pressure symptoms, and the secondary infection and draining sinuses that result from the rupture of the abscess are problems to be dreaded, although they are an important factor in the process of healing. Persistence of the abscess may have an adverse effect upon healing.[1] Spontaneous disappearance of the abscess is frequently associated with increased healing. This leads us to wonder whether surgical evacuation of the abscess has any effect on healing.

This paper is to be confined to a presentation of the treatment of the psoas abscess. The paravertebral abscess and the extradural abscess are not to be considered. The psoas abscess results from the burrowing of the detritus along the fascia of the iliopsoas muscle, which is the line of least resistance. It is of concern because of its superficial manifestation. This factor not infrequently requires differentiating the various retroperitoneal abscesses and iliac fossae abscesses of pyogenic origin. Usually, the general appearance of the patient, the difference in the acuteness of onset, and the severity of reaction lend aid in avoiding mistaking one or the other of these conditions. Another problem that arises from this tendency of the

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abscess to become superficial is pressure necrosis of the skin which may lead to a spontaneous rupture. Secondary infection is thus almost inevitable, for seldom does a hematogenous infection engraft itself in a tuberculous abscess.[2] A mixed infection takes place directly from the skin or from sinuses that result from the evacuation or spontaneous rupture. The proper and prompt care of this abscess factor is necessary if success is to be achieved in promoting healing and obviating secondary infection.

The pathogenesis of this by-product of tuberculosis of the spine has been described by many.[3] The area of degeneration making up the abscess contains detritus of the dead cells, the caseous material and necrotic trabeculae of bone. A wall of tuberculous granulation tissue lines the abscess. The continued production of debris, as evidenced by the increase in the size of the abscess indicates that the disease is progressing. As the pressure increases, from the continued production of the debris, burrowing into the surrounding tissues occurs. More than one abscess may arise from a single focus of the disease in the spine. On the other hand, the abscess may become quiescent and decrease in size; or, it may be a persistent abscess and remain indefinitely. The wall in some cases becomes calcified. Surgical evacuation of the psoas abscess is indicated when spontaneous rupture through the skin is imminent. This procedure may be necessary because of the size of the abscess.

The antibiotics are proving helpful adjuncts to surgery. There has been little tendency for the abscess to regress when streptomycin alone was used. The results have been impressive when surgical evacuation and streptomycin have been used together. The clinical improvement, the general well being, and the gain in weight have been rather striking. When febrile, the patient becomes afebrile. It has been impressive to observe the disappearance of the toxic and debilitated status. Because of the results from the use of streptomycin surgical drainage is made possible and the chance for success is much greater.[4]

Before the introduction of streptomycin, as an adjunct in the treatment of the tuberculous abscess the hazard of surgical incision was a real one. It was with hesitancy that surgery was ever instituted because of the fear of a mixed infection and the resultant continuously draining sinus tract. There was also the fear of amyloid changes that would result from persistent drainage. Aspiration with a small needle was the method of choice. Care was used to select a spot where the skin was most likely to be viable, and the puncture wound was made at an acute angle with the skin so that there might be a valve-like effect to seal off the puncture wound. One could almost say there has been a reversal of the plan of treat-

ment since the introduction of the antibiotics. Surgical incision and evacuation may be carried out with primary closure of the incised area with satisfactory results when streptomycin is used simultaneously.

It is because of this that it was felt worthwhile to write on this subject, not that it is revolutionary or unheard of, as this problem is encountered by the general surgeon as well as by the orthopaedist. Treatment frequently is of an emergency nature. Hence, the care of the abscess from the combined standpoint is a matter that is not only interesting and dramatic, but most valuable. Even though one can not as yet be certain regarding the long continuous action of streptomycin, its use is indeed impressive in the immediate healing of the abscess.

The use of streptomycin and surgery in the treatment of mixed infections and the tuberculous sinuses has shown considerable promise.[5] The healing has been dramatic. The general improvement of the patient has been gratifying. The sinuses in a large percentage of cases have healed. Incisions that have been made through the sinus tract scars have healed. This has made possible the definitive surgery which may be necessary. The usefulness of this method of handling the abscess before spontaneous evacuation and secondary infection have occurred is, thus, even more promising, for, the prevention of sinuses with mixed infection promoted more favorable healing.

When indicated, as mentioned above, surgical incision is carried out. This procedure should be done under the strictest aseptic technic. Psoas abscesses usually point in Petit's triangle, in the groin, or in the thigh region. The incision should be made in a sound area of skin a suitable distance from the "pointing spot." After the abscess is opened, the contents are removed. The cavity is flushed carefully with normal saline solution. The cavity is lined with tuberculous granulation tissue, and the direct contact of the streptomycin with this tuberculous tissue should promote healing. There should be repeated evacuations, with the usual precautions of aseptic technic and at the same time streptomycin solution should be instilled into the cavity. The supportive and general care of the patient should be attended to; rest, sunshine, proper diet and a healthy mental environment are imperative. The streptomycin may be given intramuscularly, one gram daily to adults, and graduated doses of this amount to children, depending on their age. But, when streptomycin is given intramuscularly, insufficient levels may be reached in the abscess cavity because of the poor blood supply of the tuberculous tissue.

Several ways of using streptomycin locally have been used. A

rubber catheter may be placed within the cavity and sutured to the skin. The daily instillation of streptomycin can be carried out until healing takes place, or the streptomycin solution may be instilled at the initial operation and the incision closed in layers. This latter method appears to lead to fewer sinus tracts and less secondary infection, since the catheter frequently predisposes to sinus tract formation. Streptomycin is also given systemically while care of the lesion and general condition are carried out.

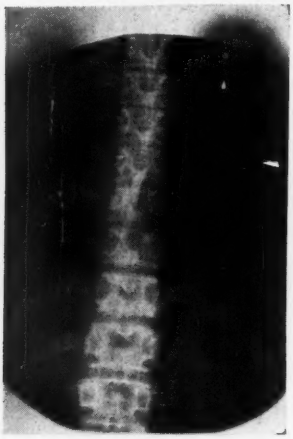

Fig. 1A

Fig. 1B

Figs. 1A and 1B. This patient had a proved tuberculous abscess. The roentgenogram disclosed no abnormality. This may well be a case of tuberculous lymphadenitis without osseous involvement. The abscess was drained and incision closed in layers after instilling streptomycin.

It is not planned here to mention the use of the Bradford frame, the plaster of paris cast and the spinal fusion as the definitive measures in tuberculous lesions.

Several cases are cited that illustrate the methods of surgical drainage and local use of streptomycin in the psoas cavity. The instilling of streptomycin solution into the cavity followed by closure in layers of the incision was the method learned while working on the orthopedic house staff of the Johns Hopkins Hospital.

CASE 1. R. F. (fig. 1), a negro male, aged 16 years, was admitted on Aug. 18, 1949, because of pains in the lower dorsal spine area of one month's duration. This pain was relieved by bed rest. During this time there had been a weight loss of eight pounds. Three weeks before admission a mass developed in the left upper lumbar region. This swollen area was not painful. The family and past history were not significant.

Physical examination disclosed a thin 16 year old colored male in no acute

distress. The temperature was 102.4 degrees and the pulse was 164 per minute. Over the upper lumbar region to the left of the dorsal spinous processes was a mass 2.5 cm. by 7.5 cm. in size and 3 cm. above skin level. The mass was very fluctuant and non-tender. There was no osseous change to palpation. There was no kyphosis or scoliosis. There was spasm of the paravertebral muscles and limited motion of this segment of the spine.

An x-ray of the lumbodorsal spine revealed no significant changes (fig. 1). The sedimentation rate was corrected to 27 mm. at the end of an hour. The

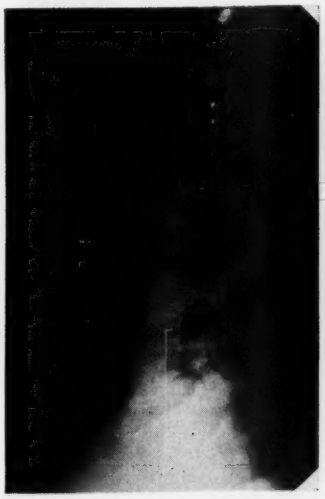

Fig. 2A

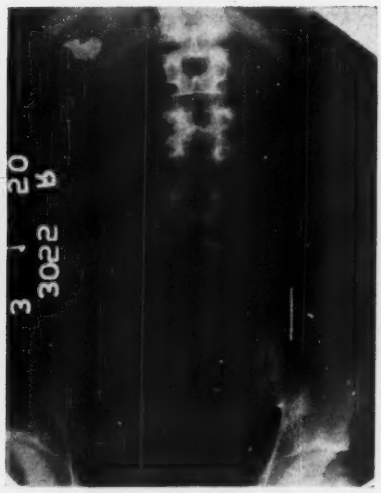

Fig. 2B

Figs. 2A and 2B. The patient with this lesion at the second and third lumbar vertebrae had a large abscess in the left inguinal region that was treated by multiple aspirations and systemic antibiotics.

hemoglobin was 11 Gm. The serology was negative. The urinalysis was essentially normal.

On August 19, an aspiration of 100 c.c. of thick greenish pus was performed at the lower pole of the mass, the culture of which was positive for the tubercle bacillus.

The patient was given streptomycin, .5 Gm., twice daily. He also was given 300,000 units of crysticillin daily.

On August 23 the abscess cavity had become distended following aspiration. On this date a surgical incision and drainage were done, with primary closure of the wound in layers after instilling 2 Gm. of streptomycin in 20 c.c. of normal saline into the cavity. Culture of material was taken at this time and was still positive for the tubercle bacillus. The biopsy of the cavity wall was reported as showing tuberculous granulation tissue. The wound healed per primum. The patient was treated in recumbency in a body jacket of plaster of paris. He was given a total of 180 Gm. of streptomycin.

The patient was last seen in the Outpatient Department on April 17, 1950.

His well being had improved strikingly. He had gained ten pounds of weight. He was afebrile. His sedimentation rate was 9 mm. at the end of the first hour. No changes were found in the x-ray of the lumbodorsal spine at this time. He remained in a plaster of paris cast. There was no evidence of a recurrence of the lumbar abscess. No sinuses had occurred.

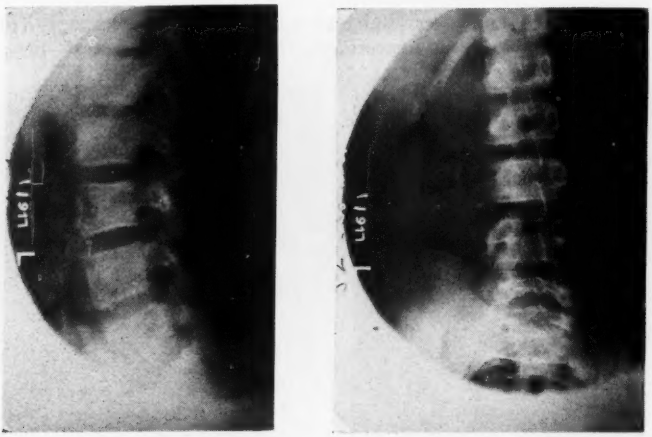

Fig. 3. The roentgenogram disclosed an early tuberculous (proved) osteomyelitis with an abscess that pointed in the left costovertebral angle.

CASE 2. H. W., a 42 year old colored male, was admitted to the hospital on Oct. 30, 1947, because of pain in the lower back. He had been unable to work on his farm for two months prior to this admission.

The physical examination disclosed a thin 42 year old man in no acute distress, temperature 98.8 degrees and pulse 98 per minute. The lumbar curve was straightened. There was tenderness over the second and third lumbar vertebral spinous processes in this region. The laboratory work was not significant. The sedimentation rate was 17 mm. in one hour. The hemoglobin was 15 Gm. The serology was negative. The x-rays of the lumbar spine disclosed destruction of the second and third lumbar vertebrae (fig. 2).

On Nov. 6, 1947, a Hibbs fusion was performed fusing dorsal twelve to sacral one. One Gm. of streptomycin was given daily. A plaster of paris body cast was worn for six months. Then this patient was allowed to be up wearing a Taylor back brace. On Aug. 11, 1948, the patient was readmitted to the hospital because of a large abscess in the left groin. This had been aspirated on July 30, 1948, and a guinea pig inoculation was positive for tuberculosis. The swelling recurred and an abscess was present at the time of admission. A second course of streptomycin was given. During this hospital admission a revision of the fusion was performed on Aug. 23, 1949. Multiple aspirations of the abscess were carried out. At the time of aspiration streptomycin solution was not instilled into the cavity. After the aspiration a sinus with open drainage occurred. This drainage has persisted for 18 months and has not closed spontaneously, even though streptomycin was given intramuscularly. He has received a total of 120 Gm. of streptomycin.

CASE 3. L. P., a 15 year old colored male, was first admitted to the hospital on Oct. 6, 1948, because of weight loss and a limp in the left hip of ten months' duration. He had a large fluctuant mass in the left flank.

Physical examination disclosed a thin colored male in no acute distress. The temperature was 100 degrees, pulse 84 per minute. There was a mass in the left flank which was fluctuant and non-tender. The laboratory work showed a hemoglobin of 10 Gm., a white cell blood count of 14,500 per cubic mm., a sedimentation rate of 14 mm., and a negative sorology.

On October 23 the abscess cavity was drained. Creamy yellowish necrotic detritus was evacuated. An erosion was seen along the third vertebral body. Two rubber catheters were inserted into the cavity and the wound was closed in layers.

The pathologic report on the biopsy from the abscess cavity was that of tuberculous granulation tissue. The tubercle bacilli were cultured from the detritus removed at operation. One Gm. of streptomycin was given daily. Daily irrigations of the tuberculous cavity were carried out, using 250 Gm. of streptomycin for each 10 c.c. of solution. This was continued for seven days. The incision healed except for a small sinus tract. This continued to drain for two months. The patient was kept in recumbency in a plaster of paris cast. His streptomycin was continued. He received in all 250 Gm. On April 7, 1949, a spinal fusion of Lumbar 1 to Lumbar 5 was performed. He was last seen on May 10, 1950. There was no drainage from the site of the drained psoas abscess and no abscess was palpable at that time.

SUMMARY

1. The importance of the care of the psoas abscess is reemphasized. A method of preventing sinus tract formation with mixed infection in handling this type of abscess is sought.

2. Several methods of using streptomycin locally in the abscess cavity are herein reviewed.

3. The method of treatment of the psoas abscess which seems to be well worthwhile is the one in which the drainage incision is closed after instilling 2 Gm. of streptomycin solution into the cavity.

4. It may be a worthwhile speculation that after incision and drainage of the abscess cavity, the systemic streptomycin is more efficacious in aiding in the healing of the osseous lesion.

5. The immediate results have been dramatic when surgical drainage and streptomycin locally have been used in conjunction with each other.

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POSTOPERATIVE ADHESIONS

A Case Report

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It is strange how some cases are indelibly stamped in the mind of a doctor, while others are seen, treated and forgotten unless a similar case reminds him. Very often the diagnosis alone is remembered along with a few snatches of the postoperative course, especially if it was stormy or eventful. Then, there is the small group in which the name, the diagnosis, the operation and convalescence are recalled in minute and vivid detail. A subdivision of this category is that group who haunt the doctor for the sheer hopelessness of the case or his own inadequacy to benefit the patient. Mrs. Jones falls into this class.

Mrs. Jones first came to me in September, 1948. She had been a charity patient for years, and had had surgery practiced on her in every sense of the word. First, it had been her appendix which had ruptured and was drained, then repair of the hernia which followed. Next her gallbladder was removed. After this, her organs of reproduction were removed piecemeal, a tube excised this time, an ovary resected the next, until finally someone, a bit bolder than the rest, made a clean sweep of the remnants of these structures. Another herniation developed and repair was attempted. She had had a vaginal plastic operation also, but the sequence of this procedure in relation to the others had long since been forgotten.

In addition to her physical discomforts, Mrs. Jones' life had been marked by tragedy. Her husband had died a violent death, leaving her a son and a daughter to care for and support. While she was a patient in the hospital following one of her operations, her son, a boy nineteen years of age, came one evening to visit her, then went home and committed suicide. Her daughter, with whom she lived, was a confirmed and uncontrollable alcoholic. At the time she came to me, Mrs. Jones was on the waiting list to be admitted to the city poorhouse.

The story of her present illness was simple, and at the same time rather typical. Although for years she had not been able to have a bowel movement without the aid of laxatives and enemas, these agents had failed three days before she came to see me. Severe abdominal cramps had started concomitantly and had continued intermittently. Nausea and vomiting followed in the wake of the

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cramps, and to use her own words "was as green as grass and bitter as bile." Although Mrs. Jones had had cramps before, they had never been so severe nor lasted so long. Also she noticed that her abdomen was swollen. She knew this to be a fact because her one good dress which she wanted to wear to the hospital would not come down beyond her chest. She was positive that she had passed no gas by rectum for three days in spite of repeated enemas.

In addition to her abdominal and pelvic difficulties, Mrs. Jones was a victim of arthritis. The disease had left its mark in the bones of her hands and spine, giving the hands a gnarled appearance and the spine a forward tilt in the cervical and upper thoracic portions. If Mrs. Jones owned false teeth, she did not wear them. As in so many edentulous persons, her cheeks were sunken and hollow, and the mandible was thrust forward and upward, bringing the lower lip nearer the tip of the nose, which was prominent and somewhat hooked. Her tongue was red and dry as tinder. Her over-all appearance reminded me very much of a Shakespearian character, or possibly the witch in Hansel and Gretel. Her neck veins were distended and transmitted the beats of her heart, which was markedly enlarged to the left side. The rate of her heart was rapid, and the action forceful and pounding as though the heart were trying to free itself from the confines of the thoracic cage. Except for coarse rales at the bases, her lungs were clear.

Everyone is familiar with the classical appearance of the caput medusae due to dilatation from stasis of the veins around the navel as seen in cirrhosis. Possibly if a patient so afflicted coughed or strained, the change in hydrostatic pressure would be sufficient to cause the veins to appear to move. I have never made such an observation personally. Mrs. Jones had a caput medusae in her lower right abdomen which was animated. Her caput was due to coils of intestines covered by thin, almost transparent, skin and scar tissue. Loops of bowel remained in constant motion either contracting or relaxing. So large was her hernial defect and so thin the covering over it that a loop of bowel could be picked up between the thumb and forefinger and held until this stimulus initiated a contraction wave which sucked the segment back into the recesses among its fellow coils. Loud rumblings could be heard plainly, and a stethoscope placed on the abdomen made the tinkles and high pitched gurgles, which were present, more audible. The rectum was devoid of feces; one more mark on the positive side of the ledger that Mrs. Jones was obstructed.

To say that Mrs. Jones was a poor risk is an understatement. She was dehydrated, out of chemical and electrolytic balance, tre-

mendously distended, and her cardiac reserve was poor. Also the time element was against her, for she stated that she had been sick for three days. Then, too, just where to look for the obstruction or obstructions presented a problem. A flat x-ray plate of the abdomen was of little help in pinpointing the offending adhesion. In a patient who has undergone eleven abdominal operations and then become obstructed, it is safe to assume that she has adhesions until proved otherwise.

I felt that the intravenous administration of glucose in Ringer's solution and intestinal intubation for decompression would be time well spent while the operating room was being prepared. The tube selected was a fairly large lumened, mercury-weighted type, for it was thought that better decompression could be gotten beyond the duodenum. Although Mrs. Jones was one of the most cooperative patients on whom I have ever passed a tube, she was one of the most difficult. Just when sufficient tube had been passed to feel certain that the rubber bag containing the mercury was in the region of the pylorus, she would open her mouth and point to the bag firmly gripped between her gums. Finally I took her to the fluoroscopic room and watched the tube enter the stomach, then double on itself and come back up the esophagus. At the suggestion of the roentgenologist, we placed her on her abdomen and tilted the table to an angle of 45 degrees. The mercury bag dropped straight to the pylorus, as though it had used a back door. Spinal anesthesia was the anesthetic of choice. Technically, it was rather difficult due to the arthritic changes about the spinous processes.

Hardly had the needle been removed and Mrs. Jones been turned on her back on the operating table, when a peculiar hissing and rumbling sound which rose and fell was heard. At the same time it was noted that her abdomen was less distended. Now it is said that one man's meat is another man's poison; also, what may be musical and melodious to one ear may be harsh and grating to the next. This combination of sounds was not in the least unpleasant to me nor to the other members of the operating team, for it indicated that something inside Mrs. Jones had relaxed, allowing gas to escape temporarily at least.

This phenomenon is not a rare one, and, as always, brought up several interesting questions. Should we proceed with the operation or should we stop and assume that the obstruction was permanently overcome? Were we justified in submitting this poor soul to another major operation, or should we send her back to her room and operate later if her abdomen swelled again? On the other hand, if the offending loop was plum-colored now, would it revert to a nor-

mal pink, or would it deepen in hue and finally become black while we were watching for distention? It was a gamble indeed, but I thought that the odds against her were less great by looking and feeling than not looking and wishing.

There were scars over the entire right side of Mrs. Jones' abdomen and from the umbilicus to the symphysis, which encroached upon the left side. There was an unscarred spot to the left of the umbilicus, a little larger than my hand. I elected to enter the abdomen through this area, hoping that the bowel and omentum had migrated and fastened themselves to the side which had received the most insults. It is said that beauty is only skin deep—what I had thought was a beautiful place to enter the abdomen was simply skin, covering a thin fibrous layer and the peritoneum. Her muscles and fascia had volatilized. Fortune was with me when I opened the peritoneum, for no bowel was attached to it where I entered. However, two inches to the right of my incision the omentum and bowel were united firmly. Her peritoneal cavity was a textbook picture of adhesions. There were short thick bands, filmy, spider-web adhesions, and violin-string bands. Loops of small bowel were matted to loops of small bowel and to large bowel. The omentum was attached to the anterior peritoneum, lateral peritoneum, and to the cul-de-sac. The transverse colon was fastened to the peritoneum as though it had been coated with glue and held until it dried. The sigmoid and descending colon were distended and multi-colored like Joseph's coat of Biblical fame. A single adhesive string ran from the left lateral gutter to the antimesenteric border of a loop of small bowel and was held taut against the mesosigmoid. The sigmoid, which was somewhat redundant and which was the only loop encountered that did not seem to be fastened down, was thickened—a probable result of flexing its muscles against this band for a number of years. When the band was clamped and cut, gas and liquid gushed and gurgled by as though they were racing to see which of the two elements would be first to get to the outside. Closure of the abdomen was effected by using multiple sutures of medium-sized alloy steel wire through the skin, alleged muscles and peritoneum. The patient was given 500 c.c. of blood during the operation.

Mrs. Jones' convalescence was far from uneventful. She ran fever, developed a urinary infection, and following a transfusion, developed pulmonary edema. I purposely waited over two weeks to remove her wire sutures because the wound became infected. I removed them then only because they cut through the skin which necrosed, necessitating grafting. Each day I trimmed a little of the necrotic skin and subcutaneous tissue away, fearing that the bowel,

which was not anchored by adhesions, would come out like a Jack-in-the box. Finally on November 2 Mrs. Jones was discharged from the hospital.

On November 30 she was readmitted with abdominal distention, nausea and vomiting, and was relieved by decompression with an intestinal intubation tube. Her red blood cell count and hemoglobin were low as well as her serum proteins. These were brought to a respectable level by the use of multiple small transfusions. She was discharged on Dec. 30, 1948, wearing a well-fitting elastic girdle which, at my request, the members of her church gave her for Christmas. I saw her every few weeks during the winter. Whenever an ambulance brought another unfortunate soul from the poorhouse to the hospital, she would come along for the ride.

Winter passed and spring came. The buds on the trees and flowers began to swell, and so did Mrs. Jones. On June 7, 1949, she was readmitted with intestinal obstruction. "Doctor," she said when I saw her, "I needs that terrible tube." Now it is well known in the minds of some patients that the vile and bitter medicine is more efficacious than the palatable. Mrs. Jones firmly believed that the intubation tube was the key to unlock her bowel whenever it became necessary. I had sowed this seed in her underprivileged mind and fertilized it well during the many long days and sleepless nights I had watched over her. Curiously enough, it worked. I would have told her anything in order to keep from going into her abdomen again!

Mrs. Jones had one more admission, her fifteenth, on June 23, before I left the city. The story was essentially the same. Each time we had been successful in decompressing her and nursing her back into a facsimile of good health.

What would cause such massive adhesions in Mrs. Jones' peritoneal cavity? Certainly it must be that some people have an inherent tendency to form them while others do not. My father referred to adhesions of this type as "internal keloids," which seems to be an apt description. We have all seen people who have been operated on once or twice or even three times with little or no formation of adhesions, or if they did, it was minimal.

Infection is known to play a role in their formation, and Mrs. Jones had a ruptured appendix. Her gallbladder was also removed, and perhaps there was spillage of bile which would set up a chemical peritonitis even if it were sterile. Glove powder has been shown to produce adhesions. With as many pairs of gloved hands as she had in her abdomen during twelve operations, it is reasonable to assume that some of the powder was left behind. Sulfanilamide powder has

also been demonstrated to form adhesions. Several of Mrs. Jones' operations were performed when sprinkling the peritoneum with this magic drug was in vogue. I am unable to state positively that this was done, but strongly suspect that it was.

The rough handling of bowel is a cause of adhesions. The odds against Mrs. Jones getting equally as gentle a surgeon with all of her operations could be figured only by an experienced bookmaker. The use of sheets or laparotomy tapes to keep the bowel up and out of the operative field may irritate the serosa of the intestine and conceivably give rise to adhesions; also the tissue can be burned by the use of such tapes which are smoking and steaming, precipitating the formation of an adhesion, for what is warm to the gloved hand is hot to the exposed bowel.

I believe that it is reasonable to assume that even when she felt her best, Mrs. Jones was in a constant state of partial obstruction. She found it necessary to whip her intestinal tract daily with laxatives and enemas to make it function. However, such use of cathartics is not too uncommon among women.

Barring an adhesive band causing a volvulus, what would cause this chronically obstructed intestinal tract to close down sufficiently to give rise to symptoms of vomiting and abdominal distention? What was the unknown factor which tipped this delicately balanced mechanism from the tolerable to the intolerable? I had a theory which seemed plausible in my own mind as a partial explanation.

In 1933 Jones and Eaton reported that edema, resulting from hypoproteinemia, was not uncommon in patients before and after operations for gastrointestinal lesions, especially those of the stomach and duodenum. Attempts to combat dehydration with sodium chloride in large amounts intensified the edema. In 1937 Mecray, Barden and Ravdin wrote concerning the effect of hypoproteinemia on the success of gastrointestinal anastomosis. They found that no matter how perfect the surgical technic, the walls of the stoma became swollen and edematous and mimicked in every way a mechanical defect in the anastomosis. However, once the serum proteins were raised to a normal level, the edema subsided and normal emptying took place. Ravdin and his coworkers also showed that the impediment to normal gastro-intestinal flow was frequently the result of an accentuation and prolongation of the edema associated with hypoproteinemia and the trauma of operation.

On several occasions Mrs. Jones' serum proteins were just below the normal figure or normal. However, she had usually been vomiting for many hours prior to admission to the hospital, and her blood was probably concentrated, giving a slightly higher value than it

would have if she were properly hydrated. Here then was a patient whose intestines were densely adherent to each other, causing all manner of kinks, bends, and abnormal twists. If edema developed in the wall of one of these sharply angulated loops, it could possibly occlude the lumen until protein was given intravenously in sufficient quantities to raise the protein concentration beyond the critical level.

The treatment she received on the four admissions following her operation was intestinal decompression, intravenous blood, plasma, glucose and amino acids. When I felt that the obstruction had passed, the patient was put on a high protein diet with supplementary vitamins. Now to ask an inmate of a poorhouse to eat a diet high in protein is as foolish as asking a rabbit to guard a head of lettuce. I realized this, and to supplement her institutional diet of bread, potatoes, and greens, or "fillers" as they are sometimes called, I collected samples of protein hydrolasates which my associates and I received in the mails. Each visit she returned to the home with a bag, full of many varieties. I believe that they helped her and cut down the number of hospital admissions to "have her batteries charged," so to speak.

What is Mrs. Jones' outlook for the future? This is such an easy question to ask, but such a difficult one to answer. It is written in the Scriptures, "He who liveth by the sword shall perish by the sword." Barring a failure of her enlarged, but good and kind heart, or a cerebrovascular accident from her elevated pressure, I fear that her adhesions shall some day be her undoing.

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A SATISFACTORY METHOD FOR DRESSING SEVERE CONTUSION LACERATED ABRASION WOUNDS OF THE FACE*

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MINOR abrasions or lacerations of the skin about the nose, cheeks or around the eyes are usually amenable to the conventional methods used in dressing wounds and are not a major treatment problem. Severe or widespread wounds usually cause more concern. To the patient, the family and friends the very thought of deforming scars frequently assumes greater importance than the patient's actual physical condition or state of health at the time of the injury.

The increasing incidence of automobile accidents has resulted in a greater number of facial and head injuries, many of them quite extensive. Such injuries are of added importance when associated with depressed or compound fractures involving the facial bones and sinuses. Industrial accidents must also be considered in discussing lesions of this nature.

Our experience in dealing with extensive single or multiple skin and faciomaxillary contusion lacerated abrasion wounds has led us to adopt a simple method of dressing which results in a most satisfying end result.

PROCEDURE

The wound is cleaned of foreign bodies such as glass, loose bone fragments, dirt, sand, blood clots and any devitalized tissue. The use of physiologic salt solution for irrigation and moist gauze sponging is preferred, care being taken not to rub or traumatize further the injured tissue. Where known sensitivity is not a factor, the wound it bathed in penicillin, 50,000 units to each cubic centimeter of solution. The wound margins are carefully approximated and maintained in position by silk sutures appropriate for the occasion, special care being taken to avoid overlapping of the skin edges or creating suture tension which will not compensate for the usual post traumatic edema. When thoroughly satisfied with the above, the extensive single or multiple abrasion sites are sponged, bathed or sprayed with penicillin solution and a flat, sterile, smooth, thin rubber sheet is placed over the entire area with a centimeter or more of free margin. Gauze is placed over the rubber and outer margin for maintaining even pressure and for absorbing any moisture from

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around the edges of the rubber sheet. The entire dressing is maintained in position with adhesive strips or bandage as desired. At the end of 72 hours the dressing is removed and reveals a granulation free surface, moist and covered with plastic exudate composed of blood elements. The sutures are removed immediately or as indicated. A similar rubber sheet dressing may be reapplied and left for 24 to 48 hours or longer as desired depending on the patient's condition (conscious or cooperative). When the dressing is removed the moist surface becomes dry within 30 minutes to several hours, it gradually hardens and forms a contracting crust which maintains the wound margins in an even position. Usually no further dressing or medication is required. The crust comes off gradually leaving a smooth new epithelial surface, devoid of extensive granulation or irregular surface. It should not be forced away from its attachments but should be permitted to drop off as it separates from the outer wound margins. Curled crust margins can be cut off with scissors.

Advantages

The method described is particularly suited for extensive abrasions or irregular contused wound skin margins. It does not require any lubricant such as petrolatum or medicated ointments to avoid sticking to the surface. This, in fact, is its main advantage. The absence of gauze mesh, which becomes embedded in the serous exudate and results in fresh bleeding when it is removed, decreases the tendency to granulation tissue formation and subsequent infection possibilities. It permits early and free removal of sutures and allows easy and repeated dressings without disturbing healing. The wound may be maintained moist as long as desired or until all the sutures are removed, thus avoiding buried sutures in crusted areas or disturbance of healing margins. The plastic exudate in the wet or dry state makes an excellent natural splint and protector for the abraided areas and wound margins. The usefulness of the rubber sheet may also be utilized in the management of burns about the face, particularly where it is necessary to avoid two moist surfaces from coming into contact such as the ears and scalp.

STREPTOMYCIN IN THE TREATMENT OF GRANULOMA INGUINALE OF THE CERVIX UTERI AND VULVA*

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A RATHER large literature describing the treatment of granuloma inguinale of the vulva with streptomycin has accumulated.[1-20] The treatment is effective, economical, and may be conducted on an ambulatory basis.[11] Whether it will be the treatment of choice remains to be determined by a comparison of results obtained with it and those obtained with aureomycin,[21-26] chloramphenicol,[25,27] and perhaps other antimicrobial agents. Conversely, the effectiveness of streptomycin in granuloma inguinale of the cervix uteri is not so well established, there being little in the literature regarding streptomycin treatment of granuloma inguinale in this site.[20,28] A possible therapeutic agent for the lesion in this location deserves documentation because here some unique and serious problems are presented. It is the purpose of this paper to discuss the use of streptomycin in granuloma inguinale of the cervix and in granuloma inguinale of the vulva, and to refer to 3 cases of the former and 5 of the latter which have been reported in a different form elsewhere.[20] In each case the diagnosis of granuloma inguinale was proved by the microscopic presence of Donovan bodies in biopsy specimens stained by the Giemsa method.[29]

CLINICAL DATA

The 3 cases of granuloma inguinale of the cervix uteri are summarized in Table I. It is pertinent to note that within two weeks following the course of streptomycin therapy all patients were asymptomatic except for a mild leukorrhea in the second case. Also of interest is the fact that no Donovan bodies were found in that part of the cervix removed by conization in the first two patients following streptomycin therapy.

The 5 cases of granuloma inguinale of the vulva are summarized in Table II. The entire group had been exposed to the usual gamut of therapy for granuloma inguinale without success. This included surgical excision,[30] antimony salts,[31] local application of such drugs as potassium permanganate, podophyllin in olive oil,[32] and boric acid. The location and size of the granulomatous lesions varied from patient to patient and ranged from small bilateral

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vulval ulcers in one instance to a lesion of the anterior vaginal wall and vulva in another patient.

Pain, which was the primary complaint in all cases, decreased

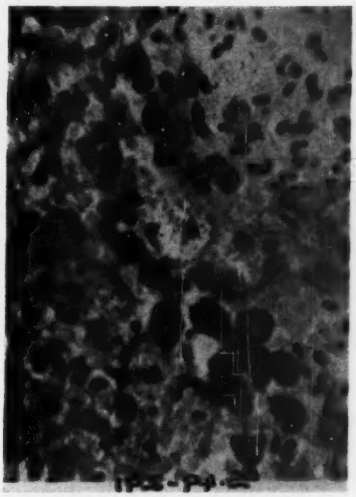

Fig. 1. Case No. 1, Patient Ju. W. Pre-treatment cervical biopsy showing intracellular Donovan bodies.

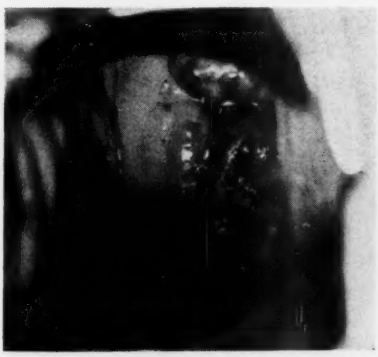

Fig. 2a. Case No. 3, Patient F. T. N. Granuloma inguinale of the cervix, pre-treatment.

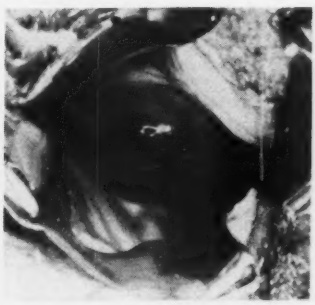

Fig. 2b. Same patient, 3 weeks following streptomycin treatment.

markedly within two days after beginning streptomycin treatment and was the initial evidence of a therapeutic response. Healing of the lesions began on the fifth or sixth day after treatment was

TABLE I

Case Summaries of Granuloma Inguinale of the Cervix

These Cases Have Been Reported Elsewhere in a Different Form[20]

Case No.	Patient	Race	Age	Duration of Disease	Gynecologic Complaints	Gross Pathologic Findings	Preliminary Impression	Pathologic Report of H&E Stained Biopsies	Pathologic Report of Giemsa Stained Biopsies	Treatment of Granuloma Inguinale	Residual Gross Pathologic Findings & Rx	Length of Follow-up	Result
1.	Ju.W	Col.	28	1 mo.	Metrorrhagia, leukorrhea	Erosion, hypertrophy, granulation, hemorrhage	Carcinoma of Cervix	Chronic Cervicitis	Granuloma Inguinale	4 gm. of streptomycin daily for 5 days	Chronic non-specific cervicitis; Conization	2 mos.	Cured
2.	A.S.	Col.	37	8 mos.	Metrorrhagia, leukorrhea, dysmenorrhea							3 mos.	Cured
3.	F.T.N.	Col.	24	2 mos.	Menometrorrhagia, dysmenorrhea						Small superficial ulcer of original disease; none	1 mo.	Cured ?

started and progressed steadily until complete epithelization occurred.

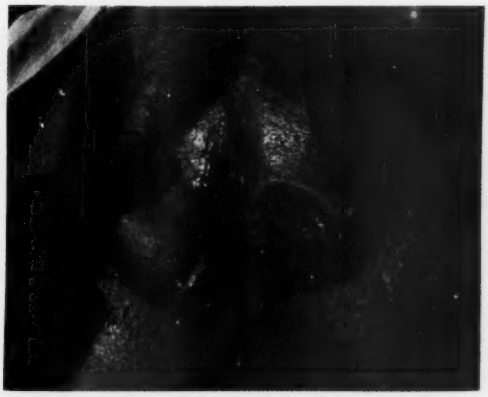

Fig. 3a. Case 4. Patient M. T. Granuloma inguinale of the vulva prior to treatment.

Discussion

The eventual treatment of granuloma inguinale will depend upon a careful evaluation of the results obtained with present and future antimicrobial agents. Much data has accumulated concerning the treatment of granuloma inguinale of the vulva with streptomycin; there can be no doubt that streptomycin is a real contribution to the treatment of this disease in this particular site. In addition, aureomycin and chloramphenicol have also been used for vulval granuloma inguinale with good results. However, data is lacking on the response to treatment, including streptomycin, of granuloma inguinale of the cervix. Available cases treated successfully should be reported because granuloma inguinale of the cervix uteri is a serious form of the disease.

Although apparently not nearly as frequent a site for granuloma inguinale as the vulva, the cervix is not infrequently involved.[20,28,33-42] Inadequate diagnostic procedures may account for its lower statistical incidence. Cervical granuloma inguinale is most frequently confused with carcinoma of the cervix;[28,37,38,43] not only are the local lesions similar, but both may produce parametrial induration with a "frozen" pelvis.[28,35,36] Since therapy for each of these conditions is now fairly specific and not interchangeable, a correct pathologic diagnosis must be made. As Speiser emphasizes, when carcinoma of the cervix is suspected but not confirmed by microscopic study, properly stained material should be examined for evidence of granuloma inguinale.[28]

TABLE II

Case Summaries of Granuloma Inguinale of the Vulva

These Cases Have Been Reported Elsewhere in a Different Form[20]

Case No.	Patient	Race	Age	Clinical Impression	Duration of Disease	Duration of streptomycin therapy	Total Dose	Length of Follow-up	Result
1.	M.L.W.	Col.	27	Granuloma Inguinale	7 yrs.	5 days	20 Gm.	10 mos.	Cured
2.	B.K.G.	Col.	21		3 mos.	5 days	20 Gm.	6 mos.	Cured
3.	A.G.	Col.	23		10 mos.	4 days	13 Gm.	10 mos.	Cured
4.	M.T.	Col.	63		11 mos.	6 days	40 Gm.	2½ mos.	Cured
5.	Ju. W.	Col.	70		5 mos.	6 days	24 Gm.	3 mos.	Cured

Cervical granuloma inguinale may be associated with severe systemic manifestations and death.[35,36] Distant bone metastasis has been described.[36,44-47] Pund and McInnes report 3 deaths in 24 cases

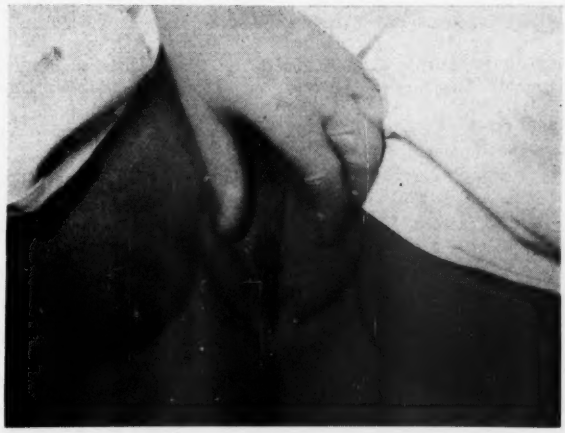

Fig. 3b. Same patient, 26 days following streptomycin treatment.

of granuloma inguinale of the cervix, a mortality of 14.3 per cent.[35] Furthermore, pregnancy complicates granuloma inguinale of the cervix. Morbidity is increased, progression of the local lesion may occur, and labor is more hazardous.[33,35-37]

A rapid and reliable method of treatment in cervical granuloma inguinale should decrease the complications in this group of cases.

Summary

1. Streptomycin has been employed in 3 patients with granuloma inguinale of the cervix uteri and 5 patients with granuloma inguinale of the vulva.

2. In each case, the diagnosis was confirmed by the microscopic presence of Donovan bodies in biopsy sections stained by the Giemsa technic.

3. Seven of the 8 patients were cured of the granuloma inguinale infection; in the eighth and most recent case, the cervix was healing satisfactorily when last seen.

4. The literature concerning the antibiotic treatment of granuloma inguinale is briefly reviewed and the unusual nature of granuloma inguinale of the cervix is emphasized.

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SOME ELECTROLYTE CONSIDERATIONS IN THE FIELD OF SURGERY*

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SINCE the advent of newer methods of measurement,[1] much attention is being paid to the study of electrolyte metabolism in the preoperative and postoperative patients. This is indeed a necessity because these patients are subjected to many procedures which tend to cause a disturbance in electrolytes. This is more likely to occur in patients with impaired cardiac or renal function, but it can be precipitated in those having good function in these organs initially. To date, there has been no routine procedure generally accepted to meet the minimal requirements, although some excellent ones have been suggested.[2] In reviewing some of the present literature, one is impressed by the possibility that the standard treatment in many hospitals may be deleterious and at times fatal. This is a rather hard concept for many to grasp as they feel that their operative mortality has been quite low over the past years, and that they have no "research" equipment to carry on necessary study.

In our experience, electrolyte disturbances are common and the resultant signs and symptoms are manifold. It is even plausible that certain unexplained postoperative deaths may have been on the basis of electrolyte imbalances. There is a very great need for the correction of these when they occur but, as Darrow has pointed out, the best method of handling electrolyte imbalances is by prevention.[3] Thus, by knowing what causes the imbalances and by treating patients expectantly, these disturbances are less apt to occur. Therefore, a more physiologic understanding is needed by many. By treating expectantly, one will have far less need for varied laboratory determinations. There are many practical points in the literature which are of help in the wiser choice of preoperative and postoperative fluid and electrolyte management. It was with these points in mind and with usual laboratory facilities that we were able to develop some insight into the cases presented.

A few of the more practical facts about electrolytes in the recent literature may now be reviewed. Much attention is being paid to potassium, but the role of sodium is also of the utmost importance.

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Recent evidences indicate that electrolytes may be lost from the body by the following chain of events:

1. When the organism is breaking down protein and is in negative nitrogen balance, the source of this protein is the cell. It has been found that accompanying this there is a loss of potassium occurring at a definite relationship, this usually being 2.8 - 4- meq/L to 1 gram of nitrogen found in the urine.[4,2] This is referred to as the potassium/nitrogen ratio and may be assumed valid in most cases of negative nitrogen balance, although some cases can lose potassium from the cell without destroying the protoplasm.[5,2] Thus, any patient losing weight, such as in an obstructing lesion of the prepylorus, will have a reduction in body potassium, even without vomiting.

2. It would seem logical that if the patient were losing endogenous potassium the kidney would conserve this ion, but Elkinton and Tarail have clearly shown that potassium will be excreted regardless of the body concentration as long as urine is being formed.[6] Thus, there is a constant excretion of potassium.

3. In situations where there is loss of gastric contents, as in vomiting and with the use of any gastric drainage, chloride is lost initially. A less amount of fixed base is lost since much of this chloride is bound with hydrogen.[7] First there is seen a depression of the serum chlorides with a beginning elevation of carbon dioxide combining power. There is also some loss of sodium early, but not in proportion to the chloride. Gamble has shown that later large quantities of sodium may be lost through the Levine tube,[8] and more recently it has been found that this is associated with considerable potassium loss.[6,9] This alkalosis after removal of gastric contents seems to be divided into two phases as far as treatment is concerned. Initially, within about the first twenty-four hours, where the loss involves chiefly sodium and chloride, saline solutions will improve the condition. After thirty-six to forty-eight hours, however, the necessary ion seems to be potassium, in which case the condition may be made worse with normal saline alone. Five per cent sodium chloride will not suffice, nor will intravenous ammonium chloride,[10] at this point. Darrow[3] has postulated an hypothesis that may hold here, and, briefly, it claims that in face of adequate renal function, when there is predominantly a loss of one ion initially, the serum carbon dioxide is inversely proportional to the concentration of potassium in the cell and is directly proportional to the cell concentration of sodium. Now if sodium were administered in the face of a potassium deficiency, it is very possible that the sodium may go into the cell.[11,3] It is also known that as sodium goes into the cell it must

carry water.[12] Thus, the physiology may be further disturbed. The results of therapy with potassium may be dramatic, and in difficult cases the falling carbon dioxide with potassium therapy may be a guide as to the amount to give.

4. Even the most minor operation in the best surgical risk will produce a reaction of the body to injury or strain, aptly termed by Selye, the "alarm reaction."[13] Selye feels that the adrenals are called into play here, and some of the recent literature confirms this impression. It is well known that the kidney, after operation, does not excrete salt normally for a period of forty-eight hours, the excretion of sodium and chloride being reduced markedly.[2,14,15] At the same time it is known that potassium is lost out of proportion to sodium during this period.[2,16] It is also known that there is a transient rise in the 17 ketosteroid excretion postoperatively.[17,2] Thus, with the retention of sodium and the increased excretion of potassium associated with the elevated cortins, this "alarm reaction" may be a very real thing. Postoperative levels of potassium where there has been adequate blood replacement may often show a lowered value reading.[2] In some cases this may be quite significant, while in others no signs result.

5. The injudicious use of saline and glucose in water may lead to significant disturbances in electrolytes. There appears to be no fixed routine in regard to fluid and electrolyte administration but in some hospitals three liters of normal saline are given daily to "well hydrate" the patient before operation. There are several objections to this, especially if further fluid is given to the average patient. In the first place, saline does not furnish "body water" unless the sodium and chloride content is extracted by the kidneys, leaving water available.[18] As the extracellular compartment is expanded, potassium is removed from the cells.[19] With the average intake of sodium for the adult being four to ten grams of sodium chloride daily, the twenty-seven grams contained in three liters of normal saline is quite in excess in the preoperative period. With the knowledge that the postoperative kidney will not excrete salt properly,[14,15] this is no longer simply an excess but at times a hazard in the postoperative period. If we consider the normal sodium of the serum to be 140 meq/L and the normal chlorides to be 100 meq/L[7] we may readily see that .9 per cent sodium chloride is hypertonic for the extracellular fluid. This contains 150 meq/L of both sodium and chloride. Thus, as this is given there are certain adjustments that the body must make, and granted the normal person may make them readily. But consider the dehydrated patient with poor renal function. It has been postulated that each three liters of normal saline will to some extent dehydrate the body cells as water will

shift to produce an osmotic equilibrium with the added slightly hypertonic electrolyte solution.[20] Also, this extra 50 meq/L of chloride must be taken care of by the kidney. This is usually accomplished by the manufacturing of ammonium,[7] and 50 meq/L is no difficulty for the normal kidney; but could not trouble ensue if the kidney had lost its ability to synthesize ammonium? Large volumes of fluid infused intravenously will tend to "wash out" body potassium.[21] There is good rationale for the administration of glucose and water but even this is not advisable when three liters of normal saline have already been given. Elman has suggested a splendid combination of both saline and glucose in water which will be discussed later.[2] Glucose will be metabolized by the liver[7] and water is a substance that is freely permeable to all body membranes.[22] Glucose is also needed to prevent ketosis.[23]

6. Draining wounds may also produce nitrogen and potassium loss.[9]

7. Diarrhea, fistulae and ileostomies,[2] and large segmental resections of the large and small gut will facilitate the loss of electrolytes and water.[24] Sodium may be lost in great quantities in the low bowel along with bicarbonate and potassium, as in part of the lower ileum the concentration of electrolytes is isotonic with the serum.[7] Here acidosis may predominate in face of a potassium deficit.[5]

With the above information one may now see why some of the newer ideas in therapy are valid and physiologic. For the routine case Elman[2] suggests 2000 c.c. of hypotonic saline with 100 grams of glucose. He reiterates Gamble's[23] observation that only 100 grams will be metabolized although some people believe that it may take 350 grams to prevent ketosis.[25] On clinical grounds and with the above facts in mind, we used a similar solution with added potassium for one year and had no severe electrolyte disturbances. This is simply made up by using a Y tube and administering 1 liter of normal saline and 1 liter of 5 per cent glucose in water at the same time. Two liters seem to meet well the minimal requirements in the average case. Our average dose of potassium was 2 grams (24 meq.) of potassium chloride in each infusion. These were given in the morning and afternoon. Oliguria must be expected on the first day, and some thirst in spite of this may be experienced, but there seemed to be adequate water and salts for body metabolism.[2]

For the complicated cases the regime will have to vary according to the depletion. With an adequate history one should be able to decide which ion is chiefly at fault, but one must also remember that seldom is only a single ion lost.

One may now ask the question: Of what consequence is potas-

sium or sodium loss? No one can answer this entirely as we are learning more daily about the various syndromes related to electrolyte disturbances. The cases presented in this paper will help demonstrate some phenomena that were of interest to the authors.

Commonly low potassium may be associated with:

1. Alkalosis.[9]
2. Weakness to paralysis.[26,27]
3. Heart failure of certain types,[28-30] possibly sudden death.
4. Mental confusion.[31]
5. Decreased renal function.[32]
6. Edema in some cases.[33,34]

Low serum sodium may be suspected in face of:

1. History of diarrhea, prolonged gastric suction, fistulae, ileostomies, and colostomies, as well as with large resections of the bowel.
2. Decreasing arterial pressure[19] to shock.[35]
3. Rising blood urea nitrogen.[35]
4. Decreased circulating protein.[19]
5. Decreased renal function.[19]
6. Edema in some cases.[36]

The electrocardiogram may be of value in detecting some abnormal concentrations of potassium in the serum,[37] but it is by no means diagnostic in all cases. Gastric alkalosis, and the alkalosis associated with over-treatment with desoxycorticosterone acetate,[38] may be a guide to the amount of potassium that should be given. It must be mentioned that at no time should the rate of administration of potassium exceed the rate of excretion as when the serum concentration rises it has a definite cardiotoxic effect[39,40] and potassium deaths are common in the literature.[41-43] If there is good renal function and at least 750 c.c. of urine per day, and if there is a cell deficit so that administered potassium will not remain in the serum; large doses may be given as needed. Oral administration is preferable, then hyperdermoclysis, and finally the intravenous route if necessary. One cannot generalize on the dose needed for a complication, but Darrow states that in children, up to .26 Gm./Kilo. of body weight may be given intravenously in a four-hour period.[3] A case of gastric alkalosis recently treated here by Dr. E. I. Evans was rapidly cured by the oral administration of 40 grams of potassium chloride daily for several days. This is not advised unless the patient can be followed very closely but may serve to demonstrate some magnitude of the deficits encountered. The most we have had to use

in severe complications is seen in Case 1. Here, in the face of severe edema and heart failure, 11.5 grams of potassium chloride given slowly intravenously as an immediate measure seemed sufficient. The entire cell deficit of potassium cannot be replaced immediately, as Butler has demonstrated that only one half of the cell potassium and one fourth of the cell phosphorus can be replaced in the repair solution.[44] Thus, it has been our principle always to undertreat initially and continue therapy over a longer period of time.

If one is dealing with a decreased serum sodium, Peters' advice is excellent. Cardiorenal cases with edema are seldom hurt by sodium chloride,[35] but they may die in pulmonary edema with the water it takes to administer it.[36] Thus, he advises the use of a hypertonic saline solution in which the maximum salt is given and the minimal water. This may be accomplished with a 3 to 5 per cent solution of sodium chloride. We have administered this to a patient with acute pulmonary edema postoperatively who had developed an inability of the kidney to hold on to chloride and probably sodium. With the history and a dangerously low serum chloride, he was given 40 grams of sodium chloride as a 5 per cent solution intravenously with dramatic recovery.

Still with all of the increasing literature, one must not neglect the looks of the patient, the tongue, skin, and the hematocrit for evidences of dehydration.

Only a discouraging word can be given for nitrogen balance. If a patient is suffering from a prepyloric obstruction from a malignancy, there is little chance of getting him in positive nitrogen balance preoperatively, although it is well worth trying to slow down the catabolism before surgery.[45] Here amino acids and protein hydrolysates[46] with added potassium may be of decided benefit, as blood and plasma will not always furnish essential amino acids. These are also available as hypotonic saline solutions with added glucose. Oral protein should be at least .3 Gm. of body weight.[47] Lockwood and Randall have recently demonstrated that regardless of the caloric intake up to and on the day of operation, it is impossible to prevent negative nitrogen balance on that day. This again may be a manifestation of the overacidity of the adrenals, and more specifically the protein catabolic hormone.[48] More recently they have been working with intravenous fat preparations.[31]

The following two cases illustrate severe electrolyte disturbances which required very special attention:*

CASE 1. (See chart 1.) This case illustrates electrolyte complications

*From the Surgical Service of Memorial Hospital, Wilmington, Del.

following surgery. B. O. was a 67 year old woman admitted because of jaundice of several days' duration. Her past history was essentially normal. Admission examination showed the blood pressure to be 190/100. The skin was icteric. There were no signs of heart failure. The liver was not felt. Laboratory studies revealed evidence of an obstructive jaundice with an icteric index of 42 units, negative stool bile, and a serum bilirubin of 6.2 mg. per cent. Gastrointestinal series suggested an enlarged pancreas. Surgery was delayed about ten days at the patient's request, during which time she was feeling well and eating normally, but also during which time her jaundice progressed with an icteric index rising to 90 units. Electrocardiogram just prior to surgery was normal. Urea clearance was 98 per cent of normal, and thymol turbidity negative.

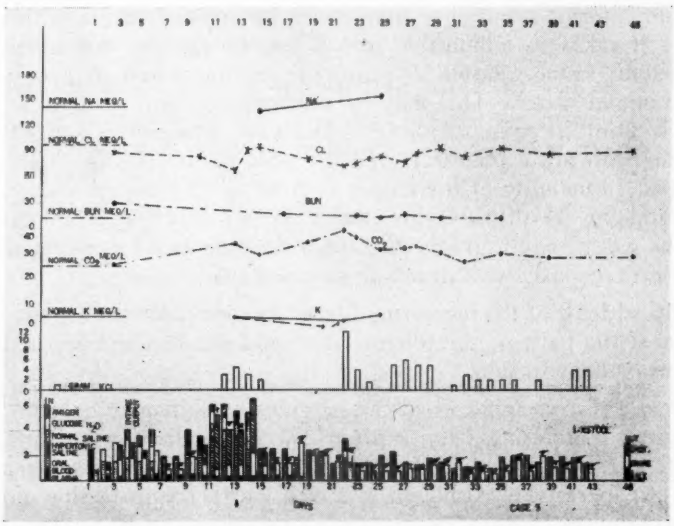

CHART 1

Her operation, about fifteen days after first being seen, consisted of a "Whipple" type procedure with resection of the head of the pancreas for carcinoma. It was thought to be completely removed. She withstood the eight hour procedure well. The immediate postoperative course was uneventful, although the bile drainage was limited in quantity. She was maintained on parenteral fluids. (See chart.) Wangensteen drainage was continuous through the first four postoperative days. Because of nausea, a Levine tube was reinserted on the ninth postoperative day. The patient was allowed unlimited quantities of fluid by mouth. A hypochlormia and hyponatremia developed, for which 5 per cent saline and small quantities of potassium were given. The potassium was ordered empirically because of her weakness and evidence of dehydration in spite of adequate fluid intake and urine output. The patient improved clinically and the Levine tube was removed several days after the concentrated saline and the potassium were given. The potassium was then discontinued. Bile was first noted in the Levine tube on the thirteenth postoperative day and in the stool on the fifteenth day. The icteric index remained elevated to about

68 units. At about this time, slight edema of the extremities was noted. Because of increasing edema, rales in the lung bases, and a persistent tachycardia, she was digitalized on the seventeenth day. It was thought that in addition to the signs of heart failure her liver function was impaired. Her serum bilirubin remained persistently elevated, being 7.5 mg. per cent on the sixteenth day. The bile drainage was always much less than expected. Mercuhydrin was given on the seventeenth day with good results but subsequent injections of 4 c.c. on the nineteenth and twenty-first days were ineffective in inducing diuresis. Dyspnea and edema became worse, and ascites were noted. The patient became weaker and confused and a congestive failure state developed with no response to digitalis, mercury or aminophylline. Although she had been eating fairly well when the mercurials were started, she refused all but sips of liquids on the twentieth and twenty-first postoperative days. Serum potassium was then found to be 3.3 meq/L and sodium 139 meq/L. She became unresponsive. Respirations were labored. Potassium chloride, 11.5 grams, were given in a twelve hour period, mostly intravenously, after which she responded dramatically with disappearance of heart failure syndrome. She was then able to sit up in bed, became oriented and once more could eat. Potassium was continued orally in doses of 2 to 4 grams per day with some added ammonium chloride. The dosage of both drugs was somewhat variable and depended largely upon the patient's willingness to take them. Her chemistries gradually returned toward normal. Electrocardiograms were taken daily during the first days of the potassium therapy in order to have warning of possible potassium intoxication. Changes from her preoperative tracing consisted chiefly of depressed S-T segments and T waves which were thought might be the result of digitalis. The tracing was not considered diagnostic of potassium deficiency but consistent with it. On the sixth day of potassium therapy the P waves became lower voltage in Leads I and II and higher in Lead III. This was interpreted as a sign of early potassium intoxication and potassium was temporarily discontinued. It was not until about the sixth postoperative week that the S-T, T wave changes in the electrocardiogram looked significantly better and this was thought to be in keeping with her general clinical improvement and increased potassium levels.

The edema and ascites slowly improved during the fifth to eighth weeks of hospitalization. Mercurials were of some help starting on her sixth postoperative week. In addition to the severe electrolyte disturbance, the convalescence was complicated by a pulmonary infarct severe enough to hazard the outcome and to require oxygen for several days. The patient's slow convalescence, particularly in strength gain, was undoubtedly complicated by liver damage. Plasma proteins which had been at a low of 4.6 Gm./100 c.c. on her twelfth postoperative day were 5.76 grams/100 c.c. with an albumin of 3.33 grams/100 c.c. and globulin of 2.16 grams/100 c.c. at the time of discharge. How much of her edema was the result of hypoprotenemia and/or portal hypertension is a matter of conjecture.

Case 1 showed several rather significant points. Gastric suction with sips of water removed much chloride as well as potassium.[6,9] This loss was also facilitated by the use of normal saline to increase the extracellular volume.[20,21] With the potassium loss continued through the urine,[6] a severe deficiency developed, as was manifested by mental depression, weakness and lethargy and loss of appetite, soon followed by heart failure.[28,30] At the point of maximum deple-

tion there was a presumptive intracellular replacement by sodium which was suggested by the rise in the serum sodium on administration of potassium alone.[3] On the seventeenth postoperative day the patient went into cardiac decompensation and failed to respond to digitalis, ammonium chloride, xanthines, or mercurials. On potassium therapy, however, the patient dramatically came out of "congestive failure-like syndrome," became alert, and was able to sit up and eat. Edema was the last complication to clear and it is noted that there were other contributory factors to the edema. It is believed that this may represent a case of heart failure associated with a potassium depletion.

The relationship of potassium depletion to edema has been noted since 1937[33] and has been reviewed recently in the literature.[34] Heart failure in the experimental animal associated with a potassium deficiency is rather well recognized.[38,49] In Case 1, the patient had a very adequate trial on the routine treatment for congestive failure and failed to show any improvement. Upon potassium therapy, however, the response was very real and dramatic. The mental depression in some cases of potassium depletion has been common in our observations, and the weakness is a well-recognized phenomenon.[26,27,50] The lack of mercurial diuresis in Case 1 may have been because mercury will not ionize to a high degree with a low chloride and a high carbon dioxide,[52] or because the kidneys had to hold the sodium preferentially in order to conserve chloride.

CASE 2. (See chart 2.) B. E. was a 51 year old obese man who developed postoperative signs of electrolyte deficiency. He was not seen by the medical service until the day of his death, when a clinical diagnosis of potassium deficiency was made in spite of uremia. Unfortunately, it was too late to institute any therapy. The admission diagnosis was carcinoma of the bladder. There was a history of slight exertional dyspnea and cough. Admission physical examination showed the patient to be well developed, well nourished and in no acute distress. Blood pressure was 130/90. Blood urea nitrogen was 19. Intravenous pyelograms showed a non-functioning right kidney. A total cystectomy was done with bilateral ureteral transplants. Because of the patient's obesity, the procedure was difficult, requiring about five and one-half hours; 1000 c.c. of glucose water and 1000 c.c. of 5 per cent glucose and saline were given during the operation. Blood pressure was maintained throughout the operation. Because of no urinary drainage in twenty-four hours, the patient was re-explored and the left ureter, which was constricted at its point of anastomosis into the colon, was reimplanted. The four hour procedure was well tolerated by the patient. There was no fall in blood pressure; 500 c.c. of whole blood and 1000 c.c. of glucose in normal saline were administered at this time. No attempt was made to isolate the right ureter. The accompanying chart illustrates the patient's fluid balance and chemical studies. He had a shaking chill with a temperature of 105 degree on his third postoperative day. Because of deep respiratory movement and weakness, the carbon dioxide was checked on the fifth postoperative day, and he was subsequently given

Ringers lactate. His course was febrile, with a temperature of 100 degrees to 101 degrees daily. An increased respiratory rate was first noted on the seventh postoperative day.

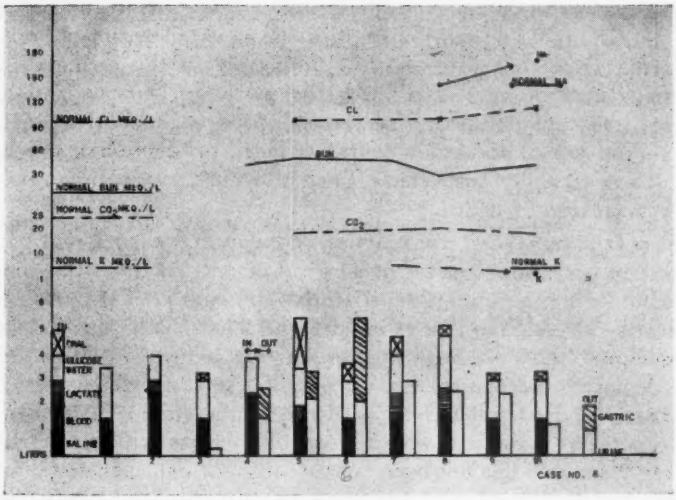

CHART 2

When seen on his last day, he appeared acidotic with respirations of forty per minute. He responded poorly. His heart sounds were fair, and the lung fields were clear. He appeared dehydrated with a dry tongue and mucous membranes, poor skin turgor, and poor muscle tone. He had been confused and irrational during the day. Weakness had been progressive for several days. Because of the clinical suspicion of potassium deficiency with an elevated blood urea nitrogen, an electrocardiogram was done before instituting any potassium therapy. This revealed depressed S-T segments in Leads I and II with a prolonged Q-T interval and low voltage T-waves which tended to confirm the impression of potassium deficiency. While an attempt was being made to give him parenteral fluids, he went into peripheral vascular collapse with associated signs of pulmonary edema and died within an hour. The serum potassium of 2.8 meq/L and sodium of 174 meq/L were only known post mortem from a blood sample taken four hours before his death.

Case 2 is of interest because one may suspect developing electrolyte deficiencies from the clinical course. This patient had neither high levels of serum potassium such as have been described for terminal uremia[53] nor hemoconcentration and decreased circulating blood volume. There was no evidence of edema except terminally in the chest. Oliguria was evident during the last day of life. In spite of high fluid intake, failure and shock did not occur except terminally when the electrolyte deficiencies were marked and fluid intake was reduced. Potassium depletion may have begun with the

adrenal alarm reaction.[13] Wangensteen drainage,[6,9] the use of normal saline,[20,21] continued urinary excretion,[6] and no attempt to replace potassium[15] all contributed to the final state. There was no striking correlation between the serum carbon dioxide and the cellular potassium.[3] The relationships of the elevated serum sodium and chloride associated with the low potassium to the terminal event perhaps may be correlated with the work of Darrow and Selye using desoxycorticosterone acetate in the experimental animal.[38,49] Again the role of potassium deficiency in the production of this heart failure is strongly suspected. Post mortem examination revealed no other cause of death.

It is felt that the alarm reaction of Selye may play a real part in initiating potassium loss in the surgical case.[13] At times this may be evoked during the operation or it may come into being during postoperative stress. Some cases not presented here have shown lowered serum potassium readings postoperatively before other factors in the depleting mechanism functioned. These cases were treated expectantly and no imbalances occurred. It is stated that when the postoperative patient is able to eat, deficiencies will correct themselves,[53] but this has not been consistently our experience.

In summary, we have indicated that imbalance of electrolytes, chiefly potassium and sodium, can give rise to serious disorders in surgical patients. Potassium loss can be expected (1) when there is protein destruction, as in undernutrition, (2) following loss of gastric contents, as in vomiting or gastric drainage of any kind, (3) under any conditions of "stress," such as an operation, (4) large volumes of parenteral fluids, particularly normal saline, and (5) draining wounds. It is also found to some degree in (6) diarrhea, (7) intestinal fistulae, (8) ileostomies, and (9) large segmental resections of the large and small gut. However, in these latter conditions the sodium loss and depletion may be of greater moment. In all these instances the kidney continues to excrete potassium as long as urine is being formed. As a consequence there is a continued loss of potassium which may lead to serious potassium deficiency. This may give rise to alkalosis, weakness, paralysis, mental confusion, edema, heart failure in some instances, and death. There may at times be electrocardiographic evidences of this deficiency.

Treatment is ideally that of prevention. That is, under the above circumstances, small doses of potassium, 50 meq. per day (4 grams of potassium chloride), should be given to prevent the development of serious deficiency. Once a deficiency has occurred, the patient should probably be given about 8 grams of potassium chloride a day if the urine output is over a liter in twenty-four hours. No rule can

be given here, however, as some cases may need forty grams per day.

While sodium is lost to some extent in all conditions where potassium is lost, it is of primary concern in (1) diarrhea, (2) intestinal fistulae, (3) ileostomies, and (4) large segmental resections of the large and small gut. Sodium deficiency gives rise to weakness, decreased arterial blood pressure, sometimes to shock level, decreased renal function with increasing nonprotein nitrogen in some cases, decreased circulating proteins, lethargy, and edema in some instances.

Sodium deficiency is prevented by replacing the lost sodium with saline solutions. Where it is marked, and particularly in the presence of edema, hypertonic saline is indicated (3 to 5 per cent).

Water depletion is treated with glucose and water solutions, 5 per cent. It has been estimated that patients need 100 to 150 c.c. of water for every 100 cal. of activity to meet all metabolic demands.[3]

Conclusions

Electrolyte considerations are of tremendous importance in the preoperative and postoperative care of surgical patients. It is probable that imbalances in electrolytes account for many of the difficulties which develop in the patient after surgical procedures and may account for a significant percentage of postoperative deaths. We have attempted to show, in a brief way, the possible mechanisms by which these imbalances might occur and how they can be managed. Emphasis is placed on expectant treatment because these conditions can be prevented in most instances if we are aware of mechanisms of their production. The mechanisms involve not only the processes of the disease but also what is being given in the way of treatment. Prevention is a much easier procedure than the treatment of an imbalance.

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PULMONARY BLEBS AND BULLAE

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THE term "pulmonary cysts" includes a wide variety of abnormal, localized distentions of the lung. Since the case report and review of the literature by Koontz[1] in 1925, numerous cases have been reported. There still exists considerable confusion as to terminology and to treatment, particularly the emphysematous blebs and bullae. The following classification of cystic diseases of the lungs as outlined by Dickson, Clagett, and McDonald[2] is a satisfactory one, and if followed eliminates confusion as to the type of cyst referred to:

I. The true developmental or so-called congenital cysts of the lungs (some of the bronchogenic cysts or bronchoalveolar cysts).

II. Acquired cysts or cyst-like cavities of the lungs.

A. "Cystic" bronchiectasis

B. Other pulmonary cysts or cyst-like cavities

1. Pneumatoceles
2. Abscess cysts
3. All other pulmonary cysts, including some bronchogenic cysts (these pulmonary cysts predominantly result from infections, trauma [penetrating wounds of chest], noxious gases, hemorrhagic effusions and other contributory factors or combination of factors)
4. Emphysematous blebs
5. Emphysematous bullae

Our discussion in this paper will be concerned primarily with groups 4 and 5, emphysematous blebs and emphysematous bullae.

An emphysematous bleb is due to rupture of the wall of an alveolus allowing air to escape into the alveolar layer of the pleura. According to Miller[3] this is due to a break into the elastic fibers of the alveolar wall allowing air to extend along the pleura in the same manner that a dissecting aneurysm extends along the wall of an

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artery. In the bulla there is a distention of the alveoli followed by rupture of the wall with formation of a distended air space. Both the bleb and bulla have a bronchial communication, although this might be quite small, particularly in the case of the bleb. They are differentiated from the true pulmonary cyst in that the latter has an epithelial lining whereas the bleb and bulla have a very thin wall formed by the alveoli. Emphysematous blebs or bullae may be single, multiple, small in size, or distended to a size sufficient to occupy the entire hemithorax. They are frequently associated with a generalized pulmonary emphysema.

The symptoms produced depend on the size, location, and the presence or absence of infection. Many produce no symptoms and are found in the course of a routine x-ray examination of the chest. There may be cough, wheezing, expectoration of blood and shortness of breath. With the formation of a check valve mechanism causing an increase in the size of the bleb or bulla, shortness of breath becomes a common symptom which may be quite marked. The condition may be confused with tuberculosis, asthma, and heart failure. The diagnosis can be established only by the use of x-ray. On physical examination the smaller blebs or bullae will be entirely missed. If they are large, absent or diminished breath sounds with associated dyspnea suggest pneumothorax.

On x-ray study the bleb or bulla is shown as an area of increased illumination with a very thin or fine line representing its wall. Fine trabeculations are usually seen transversing the cyst-like area. In cases where an entire lobe or an entire pneumothorax is involved it may be confused with pneumothorax. However, a lateral film will differentiate the two conditions. If the line of visceral pleura cannot be visualized, then a diagnosis of lungs should never be made. As a rule, in bleb or bulla occupying the entire thorax, the mass of lung tissue which is always seen at the root or mediastinal area in a large pneumothorax is not visible.

We do not feel that a diagnostic needling should ever be done in an attempt to differentiate pneumothorax from giant blebs or bullae. The danger of rupture, with fatal outcome, due to this procedure is a real one.

Case Reports

Case 1. G. T. C., white female, aged 37 years, had a routine chest x-ray in May, 1949, which showed an air-filled cyst of the left lower lobe. There were no symptoms except the complaint of intermittent mild pain in the left anterior and lateral chest for several years prior to this time. There was no dyspnea, wheezing, cough, or indigestion, and no ankle edema. The past and family histories were non-contributory.

Physical examination showed a healthy, well developed and well nourished white female with the only finding of significance being located in the left chest, where there was hyperresonance in the entire lower portion and absence

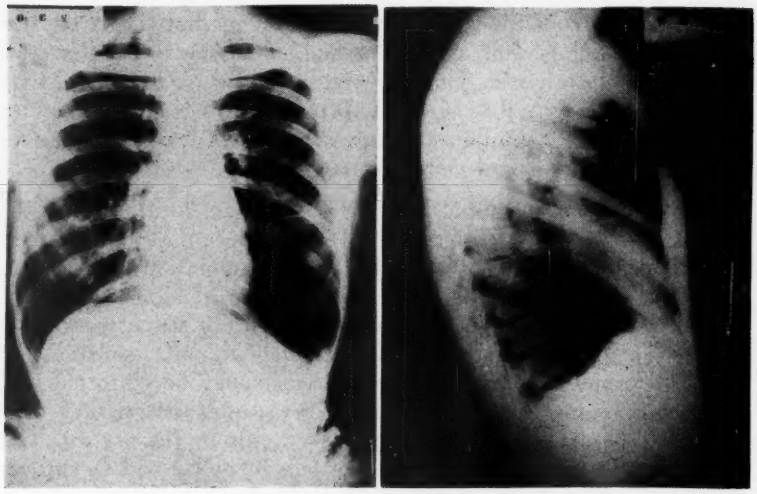

Fig. 1-A Fig. 1-B

Fig. 1. Ventral (A) and lateral (B) x-rays of Case 1 (G.T.C.). Note the large cyst in lower left lung.

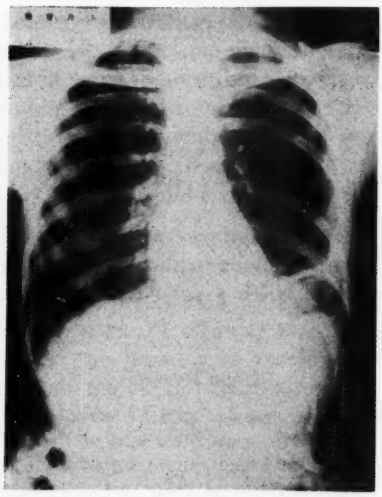

Fig. C. Postoperative x-ray essentially normal except for an adhesion about the diaphragm. The diaphragm, however, moves normally.

of breath sounds over the same area. The blood pressure was 115/70, and there was no clubbing of the fingers.

X-rays (figs. 1A and 1B) revealed a large, air filled, pulmonary cyst of the lower left lung. Laboratory findings showed a sedimentation rate of 19 mm. in one hour; red blood count 3,840,000, hemoglobin 76 per cent, white blood count 10,100 with a normal differential count. The urine was negative.

Bronchoscopy on June 22 revealed the left stem bronchus to be displaced medially and anteriorly, but was otherwise normal.

Following the bronchoscopy, a thoracotomy was done under gas-oxygen-ether intratracheal anesthesia, removing the 7th rib through a posterolateral incision. A large, thin walled, air cyst was found to occupy the entire lower third of the left thorax, and had its origin from the tip of the lingula. It was not attached to the parietal pleura, and was adherent to the anterior portion of the lower lobe by very flimsy adhesions. The cyst was opened inadvertently and no bronchial communication could be demonstrated. It was easily removed, including a very small portion of the tip of the lingula, and the lung was re-expanded. No other emphysematous blebs were seen.

The pathologist reported a large, thin walled, opened cyst which microscopically was composed of a pink staining, somewhat cellular fibrous tissue in which there were a large number of infiltrating lymphocytes with some increased vascularity. There were no lining cells other than the fibrous and granulation tissue. A small piece of lung tissue seen in the section showed some emphysema.

The postoperative course was uneventful and the patient left the hospital on the 8th day after operation. An x-ray (fig. 1C) taken 6 months later was essentially normal except that the diaphragm was still a little high, probably held in this position by an adhesion. The diaphragm was mobile on fluoroscopy.

This case showed only a large cyst in an otherwise healthy individual whose only symptom was that of mild pain in the region of the bleb. It was removed because these cysts are progressive in nature compressing much pulmonary tissue, and there is always the danger that it may rupture causing a spontaneous pneumothorax and possibly an acutely ill patient. It is interesting to note that the pathologist reported a moderate amount of chronic inflammation of the cyst wall even though no infection was seen grossly. The anemia which was noted preoperatively (hemoglobin 76 per cent) might possibly be explained on the chronic infection, since no other cause was found.

Case 2. E. E. H., a white married female, aged 28 years, complained of moderate progressive dyspnea for several months, with no severe chest pain, no hemoptysis, and no ankle edema. She stated that she had had bronchial trouble for six years, accompanied by a mild cough and considerable wheezing in her chest whenever she had a cold. An x-ray taken five years prior to this time had showed only healed scars of tuberculosis (fig. 2). The past and family histories were non-contributory except for a sister and a brother who had tuberculosis.

The physical examination was negative except for the chest where there was hyperresonance over the entire upper right thorax, and suppression of breath sounds posteriorly from the 4th to the 7th rib. No wheezing was heard. The blood pressure was 115/70, and there was no clubbing of the fingers. X-ray examination (figs. 2B and 2C) showed a large, thin walled, air filled cyst on the right side occupying the upper two-thirds of the thorax. Laboratory data revealed a sedimentation rate of 13 mm. in one hour, hemoglobin 90 per cent, red blood count 4,190,000, white count 11,000 with a normal differential count. The urine was normal.

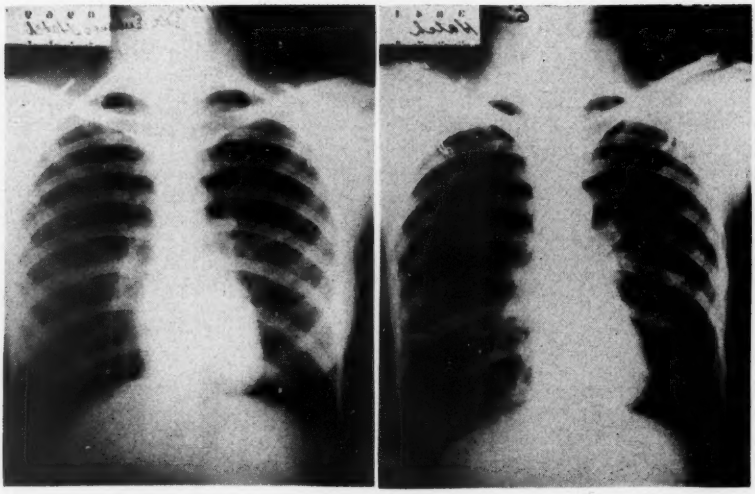

Fig. 2-A Fig. 2-B

Fig. 2. (A) Roentgenogram of Case 2 (E.E.H.) in 1944 showing no cyst.
Fig. 2. (B) See figure 2-C.

Thoracotomy was performed on Nov. 11, 1949, under gas-oxygen-ether intratracheal anesthesia, resecting the right 6th rib through a posterolateral incision. A very large, thin walled, air filled cyst was found to occupy the upper two-thirds of the right pleural cavity, which had its origin from the lower portion of the upper lobe posteriorly. No bronchial communication could be demonstrated and the cyst was easily removed and the lung re-expanded. No other emphysematous blebs were seen.

The pathologist reported a large thin walled cyst previously opened, and microscopically showing the wall to be lined by mesothelial-like cells. A few infiltrating plasma cells and lymphocytes were present.

The postoperative course was uneventful, and the patient left the hospital eleven days after operation. Recently she was seen because of acute pharyngitis which subsided promptly, and she stated that she has been considerably improved by surgery.

An x-ray (fig. 2D) taken six months later was essentially normal.

This patient had more symptoms than Case 1 in that she had

had some progressive dyspnea for a few months. Removal of the cyst allowed full expansion of the remainder of the lung, and complete relief of symptoms was obtained. The prognosis should be excellent because of the fact that no other blebs were noted in this lung. She had a normal roentgenogram in 1944, hence the bleb has developed during the past five years. It is interesting to note that in her history she stated that she had had some bronchitis or

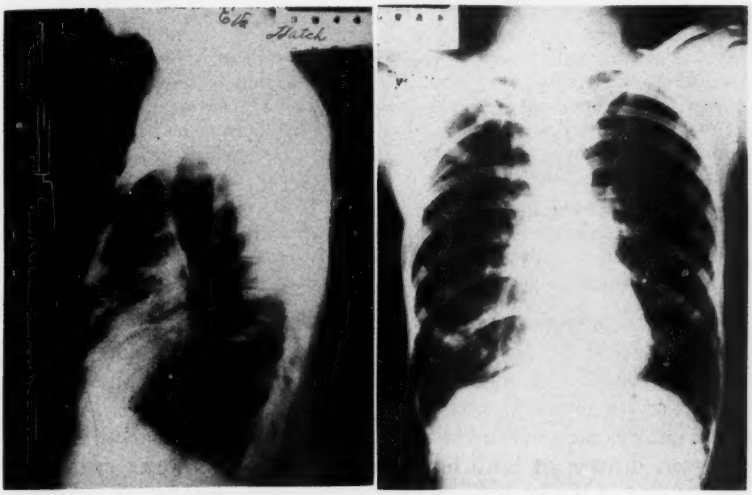

Fig. 2-C Fig. 2-D

Figs. B & C demonstrate large cyst in upper right chest on ventral and lateral films.
Fig. D. X-ray taken 6 months postoperatively is negative.

bronchial trouble since 1943, thus possibly indicating that infection plays a role in producing these blebs. Also the pathologist reported little evidence of inflammatory process in the cyst and she had no anemia as compared to Case 1.

CASE 3. A. E. L., aged 43 years, white married male, gave a history of shortness of breath on exertion for 3 years, which was progressively becoming worse, and without ankle edema or chest pain. He had recently lost his job as a streetcar motorman because of dyspnea and was working as a part-time janitor. There was a chronic cough, productive of a white frothy sputum, occasionally purulent, and he had at times expectorated a small amount of blood.

Physical examination on Nov. 6, 1941, in the outpatient department of the Medical College of Virginia, was essentially negative except for dyspnea on even slight exertion, an emphysematous appearing chest, generalized hyperresonance, and distant breath sounds bilaterally. He was afebrile and had a blood pressure of 150/108. Venous pressure and circulation time studies were normal. The vital capacity was 37 per cent of normal. There was a persistent

mild polycythemia, and the remaining laboratory studies were normal including the urinalysis.

X-rays (films have been destroyed) showed an emphysematous type of thorax with a large air filled cyst in the upper right chest which on light exposure had a suggestion of fine trabeculations in the cavity.

On November 28 a needle was inserted into the cyst to obtain pressure studies which were found to be plus 10 and minus 8. Shortly after the needle was withdrawn he developed severe right chest pain and became intensely dyspneic. He was hospitalized at the Medical College of Virginia Hospital and an intercostal catheter was inserted and connected to underwater drainage. He continuously lost air through the tube with each expiration until he died. Continuous nasal oxygen was administered, but in spite of this he still showed some cyanosis and extreme dyspnea. He was never an operative risk and steadily declined and died on Dec. 27, 1941. During his last two weeks, the non-protein nitrogen determinations rose to 100, which was attributed to renal damage resulting from sulfathiazole therapy.

Autopsy showed multiple emphysematous bullae of varying sizes in both lungs, and a larger bleb which had been ruptured in the upper right lung and which had an easily demonstrable bronchial communication. There was a considerable compression of the right lung resulting from the pneumothorax.

This case demonstrated chiefly the danger of aspirating an emphysematous cyst. The opening through which the needle was inserted never healed, and probably because of a ball-valve action, the intracystic pressure was too high for healing to occur. As demonstrated by autopsy and also clinically, he had extensive emphysematous disease of both lungs, and the pneumothorax caused by perforation of the larger cyst so decreased the amount of available pulmonary tissue, in a patient with little or no reserve, that he could not compensate for it and died.

The case also demonstrates the type of patient who would probably not be relieved by surgery because of the extensive bilateral disease. Had spirometric studies been done, they would probably have shown tremendous impairment in the function of both lungs, and hence a very poor operative risk, and the benefits that would be obtained by removing the larger cyst would be very doubtful. Such severe cases as this should probably be treated medically with emphasis on controlling the infection, bronchiospasm, and allergy.

Case 4. G. L. B., white male, aged 38 years, had a stenosis of the right middle lobe bronchus resulting from a tuberculous infection. A thoracotomy was done for lobectomy, and an incidental finding was that of a solitary, moderately large, somewhat pedunculated emphysematous bleb, about the size of a large orange and arising from the extreme upper portion of the upper lobe. It was easily removed, and no bronchial communication was discovered. This bleb could not be demonstrated on x-ray (figs. 3A and 3B).

This case is presented only to show that many emphysematous blebs cannot be demonstrated by the roentgenogram. Even after

thoracotomy had shown the above cyst, we could not demonstrate it on reviewing the films. Burnett[4] and others have stressed this point, and because of this it seems that spirometric studies, which we have not used, to determine the individual function of each lung should be most helpful in selecting cases for surgery.

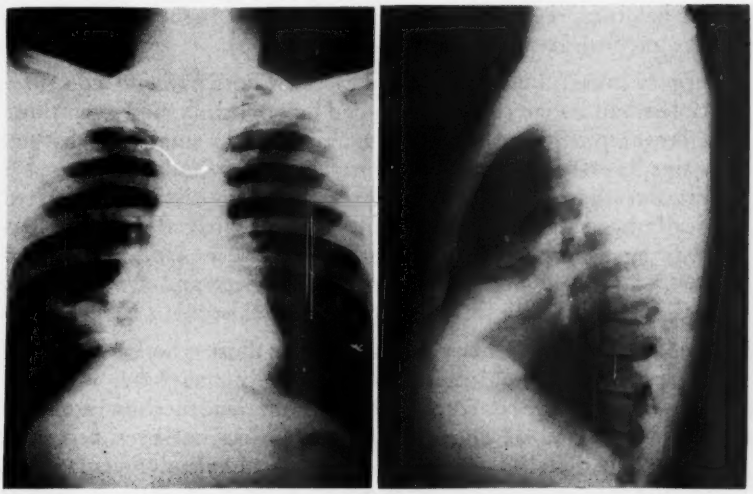

Fig. 3-A Fig. 3-B

Fig. 3. (A) and (B) of Case 4 (G.L.B.) demonstrate no cyst in upper right lung which was shown to be present by thoracotomy performed for middle lobe lobectomy for tuberculosis.

COMMENTS

In commenting on this type of pulmonary disease, we state again that its cause is not exactly known, but is probably the result of infection, or allergy, or bronchiospasm or a combination of any of these. It seems quite likely that some type of ball-valve mechanism is present at some time or other with resulting increased intrabronchial pressure causing a balloon cyst which progresses in size. In our opinion, these cysts or blebs should never be aspirated, either to remove air or to measure intracystic pressure, because of possible fatal effect as shown in Case 3. Many have been needled with no harmful effects, but they most probably had no large bronchial communication and a ball-valve mechanism. We know of no way to determine preoperatively which of these cysts have such a communication, and have strictly abstained from needling since 1941. Intracavity studies do not aid in making the diagnosis. In fact the diagnosis can easily be made from the roentgenogram alone, in

that it shows a typical rarified area with occasional fine trabeculations in the cavity on light exposure, and usually a portion of the thin wall can also be seen. It is recognized, and recently pointed out anew by Chamberlain[4] that the smaller blebs or cysts frequently do not even show on x-ray as demonstrated in Case 4. However, the large cyst can easily be seen on the roentgenogram due to the fact that it has compressed the adjacent pulmonary tissue, and presents a large rarified area.

Surgery is useful only in selected cases. Those patients who show multiple small bullae cannot be helped by operation, and even those with large cysts, who have additional multiple smaller blebs and extensive fibrosis of the lung, can rarely ever be helped. This is a progressive disease, and the latter two groups should be treated medically by eradicating infection, correcting their allergies, relieving their bronchospasm, and by antibiotic therapy. Under this treatment, many patients show clinical improvement and a few have a partial or even complete arrest of their disease.

Cases which respond best to surgery are those who have a single large cyst, or multiple cysts involving only one lobe, or those patients who have bilateral cysts without evidence of extensive smaller bullae and/or fibrosis. Lobectomy has not been necessary in any of our cases, and it seems that it would rarely ever be indicated, for they usually can be removed easily with little or no loss of pulmonary tissue. It seems most important in emphysematous disease to preserve as much pulmonary tissue as possible, leaving such procedures as lobectomy or segmental lobectomy to the treatment of true cysts of the lung.

The large solitary cysts should be removed surgically in order to prevent rupture with resulting spontaneous pneumothorax, if there is a large enough bronchial communication and a ball-valve mechanism. Removal of these large cysts also allows re-expansion of collapsed lung adjacent to the bleb, and thus reestablishes full pulmonary function.

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MULTIPLE METASTASIZING CARCINOIDS OF THE ILEUM WITH SIMULTANEOUS PAPILLARY ADENOMAS OF THE COLON COMPLICATED BY INTUSSUSCEPTION AND GANGRENE

Case Report with Operation and Recovery

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THE incidence of carcinoid tumor is highest in the vermiform appendix and much lower in the cecum, small bowel, and stomach. In the former it usually produces symptoms referable to the appendix whereas in the latter the tumor may spread and metastasize without drawing attention to its presence. However, the relatively few cases reported in which the presence of such tumors necessitated surgical intervention encouraged us to report our case.

This case presents a number of interesting features, namely, (a) multiple papillary adenomas of the colon; (b) multiple metastasizing carcinoids of the ileum, the most distal of which caused (c) ileocecal intussusception and gangrene; (d) operative removal, prolonged convalescence, and recovery in a woman seventy-one years old.

CASE REPORT

B65340 V. K. T., a colored female, 71 years old, was admitted to Saint Philip Hospital on Feb. 10, 1949, with the chief complaints of a lump in the stomach for ten days, and pain and vomiting for the thirty-six hours preceding entry.

Previous History: A duodenal ulcer was diagnosed in 1931 for which a posterior gastroenterostomy was done in 1932, at Saint Philip Hospital, after which symptoms of peptic ulcer disappeared. No reference was made regarding the presence or absence of intestinal tumors at the time of laparotomy.

In 1944 she developed mild diabetes mellitus which was controlled by diet.

Findings on Admission: A cooperative, elderly, moderately well nourished, colored female, with temperature of 99 F., pulse rate 90 per minute, blood pressure 220/110. The heart was enlarged, and there was a blowing systolic murmur over the precordium. A firm, freely movable, non-tender 10 by 20 cm. mass was found in the right lower quadrant. Rectal and pelvic examinations were negative.

The only significant positive laboratory findings were a fasting blood sugar of 154 mg. per cent, urine sugar 3 plus, and urine albumin 1 plus.

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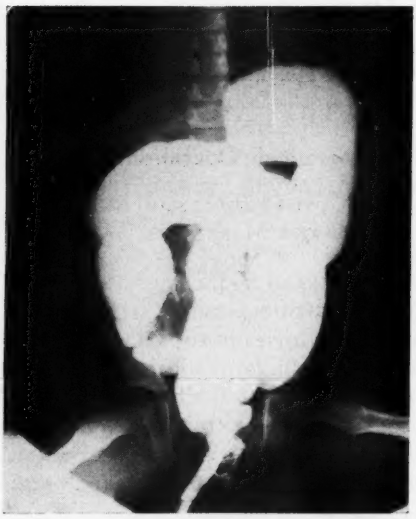
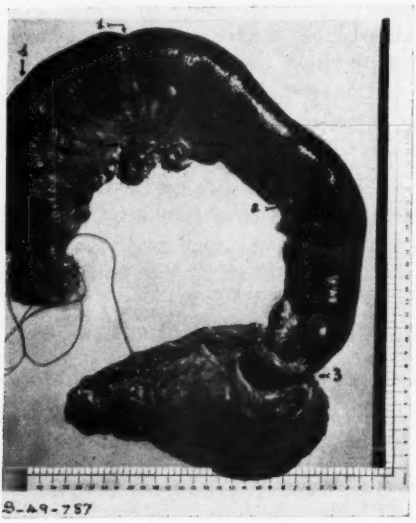
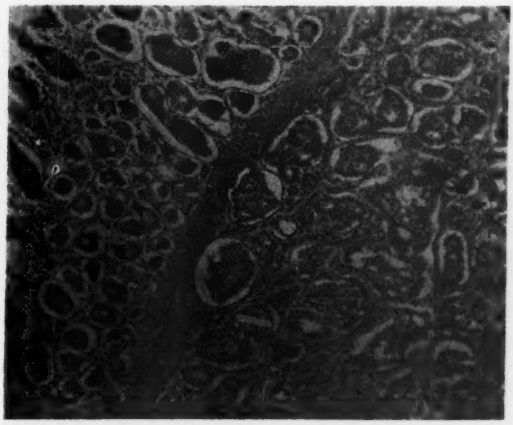


Fig. 1. Roentgenogram 49-2162, showing barium filled colon with site of lesion in the ileocecal region.



Photograph of ileum and colon showing: 1. The primary carcinoid lesions. 2. The enlarged mesenteric lymph nodes. 3. The site of the intussusception.

On February 12, roentgenologic examination following a barium enema was suggestive of extrinsic pressure on the cecum (fig. 1). Immediately following the barium enema she developed signs of severe intestinal obstruction.



Photomicrograph 1. Hematoxylin-eosin x 200. Showing solid and adenomatous foci of the carcinoid tumor in the mucosa and submucosa.

This was partially relieved by the use of a Miller-Abbott tube. Her condition fluctuated, and four days later an exploratory laparotomy was performed.

Operative Findings: The peritoneal cavity contained about 1500 c.c. of clear straw colored fluid. The distal end of the ileum contained indurated whitish nodules with firm mesenteric nodes which on frozen section revealed adenocarcinoma. There was also ileocecal intussusception and a mass about 20 cm. in length. Ileocolectomy with side to side anastomosis of the ileum to transverse colon was performed.

The postoperative course: A wound infection and a small fecal fistula developed which, after prolonged convalescence, closed spontaneously. At the present time (thirteen months after surgery) the patient is well.

The Surgical Specimen: (S49-787.) Gross Description: A piece of bowel which includes 30 cm. of terminal ileum continuous with 20 cm. of the ascending colon. There is a cuff of ileum which is very thick and forms a distinct ring through which the small bowel intussuscepts. The appendix forms a part of the cuff. Along the antimesenteric border of the terminal ileum there are three discrete, indurated, puckered, yellowish-white areas which protrude towards the lumen. The ileomesenteric lymph nodes are enlarged and firm (fig. 2).

On opening the colon, the invaginated loop of bowel measures 22 cm. in length. The distal 4 cm. of this mass is gangrenous. On the medial aspect of the proximal portion of the ascending colon, 3 cm. distal to the cuff, there are two sessile polyps, one measuring 3.5 cm. and the other 1.5 cm. in diameter; at 11 cm. and 15 cm. two other pedunculated polyps are found, each measuring 2 cm. in diameter.

On opening the telescoped ileum, a large soft polypoid gangrenous mass 3 by 1.5 cm. is found. This mass is apparently the cause of the invagination.

Microscopic Findings: Sections through the tumors in the ileum show carcinoid tumors infiltrating the mucosa, submucosa, muscularis, (Micro 1)

Photomicrograph 2. Hematoxylin-eosin x 60, showing carcinoid metastasis in a lymph node.

Photomicrograph 3, showing normal mucosa of cecum and papillary adenoma.

serosa, and the regional lymph nodes (Micro 2). The tumor is composed of solid nests and the alveolar structures with lumina, in which the cells are uniform in size and shape. No mitotic figures were seen. In some of the cells the cytoplasm contains fine chromaffine granules, in others the cytoplasm stains a homogeneous orange-yellow color; and in others the chromaffine reaction is absent. Those findings are noted in all the tumor nodules. The gangrenous portion of the ileum shows ghosts of carcinoid nests. Neither the appendix nor the colon contain carcinoid foci. Sections of the cecum showed benign papillary adenomata (Micro 3).

Discussion

A brief review of the history of carcinoid tumors in general may help in understanding their nature and clinical behavior when they occur in organs other than the vermiform appendix. Merling (1838)[1] was the first to report on a primary carcinomatous-like growth of the appendix. Langhans (1867)[2] described a tumor involving the submucosa and muscularis of the ileum and presenting alveolar and infiltrating characteristics. Lubarsch (1888)[1,2] was the first to differentiate carcinoid tumors from ordinary adenocarcinomas. Oberndorfer (1907)[1,2] suggested the term carcinoid tumors to denote their benignancy. However, Ranson (1890)[3] reported a case of carcinoid of the ileum with metastases to the mesentery and liver. Thereafter these infiltrating growths have been reported in the stomach, small intestine, vermiform appendix, and colon; but mortality, resulting from the tumor per se, has been rarely encountered.

The Histogenesis of Carcinoids: Different theories have been postulated among which are the following: (a) that they are analagous to basal cell carcinoma of the skin; (b) Cohnheim's theory of embryonic rests akin to cells of pancreatic islets; (c) Aschoff considered them gastrointestinal nevi; (d) Foot and Minckler both include them among the miscellaneous peripheral neurogenic tumors although (e) Masson (1914-1930) had already demonstrated their origin from the argentaffine cells in the crypts of Lieberkühn and proposed the term neurocrine and argentaffine cell tumors.[1,2,4-10]

The Incidence of Localization of the carcinoid tumors is highest in the appendix, the ileum next, while the stomach, duodenum, jejunum and rectum are rarely involved; 85 to 90 per cent of carcinoid tumors occur in the region of the ileocecal valve.[2,4,7]

Carcinoid tumors are encountered in approximately 0.2 to 0.5 per cent of surgically removed appendices. Their presence may be responsible for distention or obstruction with the resulting appendicitis symptoms. The average age is 25 years.[2]

Etiology of the Symptoms: Masson (1924-1928)[2,5] demonstrated neuromata rich in argentaffine cells in obliterated appendices and originated the neurogenic theory of carcinoid chromaffine (Kulchitzky) cells of the crypts of Lieberkühn. Porter and Whelan (1939)[7] considered chronic inflammation as a predisposing factor in the development of appendiceal carcinoids. However, in carcinoids of other organs which lead to surgical intervention, an inflammatory process in the neighborhood of the tumor was found responsible for the symptoms. Similar findings led Dockerty and Ashburn (1943)[6] to consider carcinoids as adenocarcinoma grade I,

because their course and symptomatology, and complications are similar to those of slowly growing adenocarcinomas occurring elsewhere in the gastro-intestinal tract. Whatever the causative factors covering the origin of this neoplasm (whether embryonal rest, neurogenic, or epithelial) and not disregarding recent literature which stresses its malignant aspects, the uniformity of its cells, its chromaffinity and argentaffinity, its consistently mild course (with few exceptions) in spite of widespread infiltration and metastases,[4,5,11-17] we feel that the long established term "carcinoid," though incorrect, is more expressive of its nature. To consider it a grade I adenocarcinoma implies that there are all gradations to more anaplastic forms. However, the latter implication could not be established, as all literature agrees that present technics are inadequate in differentiating the localized from the invasive and malignant forms.

Clinical Applications: In most instances in which symptoms producing carcinoids have been encountered during surgery, the operative procedures were those of necessity or emergency nature. In spite of recent advances in therapy and technic, radical intestinal resections in "unprepared patients" still presents significant morbidity, complications, and prolonged convalescence.

Chronic recurrent intestinal obstruction, or indefinite appendicitis must be completely evaluated for the possible occurrence of an ileal carcinoid.[18] Roentgenologic attention to small bowel patterns for kinks or filling defects,[19] and adequate exposure and examination of cecum and ileum must be stressed during the operative procedures in this group.[18,20]

Surgical resection in the presence of infiltration and metastases is the treatment of choice, with resulting good prognosis.[9,18] However, the cases are recorded in which grossly inoperable carcinoids were by-passed with a simple ileocolostomy and the patients survived ten and fifteen years following operation. Death from other causes proved by autopsies showed that the tumor though present did not increase in size or cause incompatible damage to the organs to which they had metastasized.[6,21]

Although the tumor had been reported to melt[4] on irradiation therapy, it is an unwise procedure without previous adequate exploration.

The simultaneous occurrence of carcinoids and other tumors of the intestinal tract have been reported only in few instances. Morgan[22] reported an adenocarcinoma of the cecum, and the incidental findings of a small carcinoid in the distal ileum; Ariel[4] reported a case of adenocarcinoma of the colon, benign polyposis of the colon and an ileocecal carcinoid tumor. The presence of one had camou-

flaged the existence of the others and a resulting intestinal obstruction had been the main symptom-producing factor.

It is difficult to establish in the present case whether or not the tumor led to inflammation, or the inflammation to tumor. Carcinoids being of neurogenic nature may be indicative of perverse neuromuscular peristaltic activity. However, inflammation and a protruding mass in the mucosa has been shown to cause invaginations of the bowel. It is noteworthy that following the barium enema in our case, the patient developed more severe symptoms of intestinal obstruction, which on operation proved to be a gangrenous invagination with one of the carcinoid polyps as the spearhead.

The intussusception of the small carcinoid in Morgan's case was a non-obstructive "small, beginning ileo-ileal intussusception." The only description of an obstructive invagination that we could find in the literature occurred in Ariel's case in which other simultaneous colon neoplasms were present.

The case we present is the only one in which gangrene of the bowel resulted from intussusception and strangulation of a polypoid carcinoid of the ileum.

SUMMARY

1. A case of intussusception with gangrene complicating multiple carcinoid tumors of the ileum and multiple papillary adenomas of the colon in a 71 year old colored woman is presented, with operative removal and recovery.

2. The history and etiology of carcinoids are briefly reviewed.

3. Owing to the absence of anaplastic gradation in carcinoid tumors we prefer the use of the term "carcinoid tumors" to that of adenocarcinoma grade I.

4. Clinical consideration of diagnosis and treatment of carcinoids in general are discussed.

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OSTEOMA OF THE CRANIAL VAULT AND BASE

Three Cases Illustrating Two Varieties

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Richmond

DIFFUSE osteomata of the cranium are most frequent in the region of the frontal and mastoid sinuses, as stated by Dandy.[1] This report summarizes our experience with three recent cases, all seen in 1949. Two of the patients (both young children) had diffuse bony tumors involving the frontal bone, both of the vault and the base; these tumors were dealt with by rongeuring away the bone piecemeal, including the roof of the orbit or orbits, as well as the anterior fossa itself, which was greatly thickened in each instance as far posteriorly as the sphenoidal ridge and the anterior clinoids. A tantalum plate was inserted in each instance to restore the continuity of the anterior cranial vault. The third patient (an adult) had an osteoma arising in the right temporal bone. It involved the vault of the skull as well as the base. It was removed by rongeuring bone away rather widely around the osteoma, after which the dura was opened completely around the bony tumor and involved skull. The entire mass of skull and dura, as well as the tumor itself, was removed en bloc precisely as one removes a meningioma of the cerebral convexity which has grossly invaded the overlying dura and skull. The mastoid cells were entered in the necessary mobilization of the tumor. They were immediately closed with fibrin foam and no subsequent infection ensued. The bony tumor closely approximated the adjacent lateral and, especially, the sigmoid sinus, which, fortunately, were not opened during the dural section just before removal of the tumor which had deeply indented the underlying temporal lobe and had thus caused a clear-cut contralateral homonymous hemianopsia. Uninterrupted recovery occurred in each of the three patients.

Diffuse osteomata in the frontal region, the most common site, are frequently bilateral. They merge into normal bone, though thickened osteomatous bone in transition to the normal may cover a large part of the vault of the skull. The growth is both inside the cranial chamber and on the outside. The x-ray shadow is characteristic and extremely dense. The paranasal air sinuses or, in temporal bone lesions, the mastoid, may be grossly involved.

Ford[2] states that some of the osteomas in childhood are merely

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small endostoses which arise in the base, often from the lesser wing of the sphenoid, the clinoid processes or the floor of the middle fossa. There are also massive tumors of the vault. The first two cases to be described in this paper (Cases 1 and 2) are examples of a combination of these two types of osteomas. Ford[2] also clearly distinguishes between the above described tumors and those which arise in the frontal and ethmoid sinuses.

These tumors are, of course, usually benign histologically, but may be termed malignant by position, particularly because of their *basilar* location. If this portion of the growth is not completely removed, often a difficult task because of encirclement of important nerves and vessels, slow but inexorable growth and further thickening of bone is liable to continue. We have reliable information of one such tumor (illustrating the hazards of removal) operated upon in another clinic. It was an osteoma with gross and widespread thickening of the floor of the anterior fossa: in manipulation of the bony mass incident to its being rongeured away, the internal carotid artery was inadvertently torn, with immediate fatality from uncontrollable hemorrhage. The third (temporal bone involvement) case to be reported in this paper had, fortunately, only slight involvement of the base and presented a quite well-defined border within the cranial chamber. Usually, however, they merge gradually into normal bone, especially those located anteriorly in children, and involve the roof of the orbit on one or both sides and may fill one or both frontal sinuses, causing a diffuse protuberance externally (figs. 1 and 4). At other times, there is a tremendous diffuse thickening of the skull. It may cover a large part of the skull and thus demand surgical removal because of the reduction in size of the cranial chamber, causing severe headaches and, at times, as in the temporal lobe case reported in this paper, focal signs and symptoms. The removal of these osteomata is frequently a sanguinous procedure and may have to be done in two stages because there is an elaborate blood supply derived from both the dura and scalp. For diffuse bony growths, it is necessary to make numerous burr openings around the periphery when this is possible or feasible. These may be joined by cutting with the deVilbiss rongeur, or, in the deeper parts, the openings are joined with large bone-cutting instruments. It may be impossible to remove the outer *basilar* limits of such growths because they grow into the frontal sinuses and frequently also involve the roof of the orbit and the anterior fossa but they grow very slowly and even in such instances, operation is productive of very satisfactory results. It may be necessary to remove the walls of the optic foramina (especially if visual acuity is diminished), the anterior clinoid processes, and a considerable portion of the sphenoid wing or wings. Either

immediately after the osteoma has been removed or after subsequent x-ray films have demonstrated the complete removal of the tumor, it is advisable to insert a tantalum plate to cover the defect in the skull vault, as was done in each of the three cases reported in this paper.

The two cases of diffuse frontal osteomata presented in this paper are not to be confused with the other well-known type in this region: those bony tumors *arising* in the paranasal air sinuses (especially the frontal and ethmoid sinuses) and which are *rather discrete masses* which frequently erode through the dura and directly depress the anterior pole of the frontal lobe. These are best attacked by a frontal osteoplastic flap, depressing or actually opening the dura over the frontal lobe and eventually by gently "rocking" the tumor from its bony bed in the orbital roof, the ethmoid or frontal cells, and the anterior fossa itself. Such tumors begin in the *sinuses*, and the mucous membrane of the sinus becomes tangled about the bony nodules with the production of numerous small mucoceles. The appearance is often that of a bunch of grapes.

Cushing,[3] in 1927, reported four cases of pedunculated orbito-ethmoidal osteomata in his Presidential Address read before the American Surgical Association, but these were primarily pedunculated osteomata of the ethmoid cells which secondarily came to involve the orbit. One of his cases had a huge intracranial pneumatocele as a complication of an orbito-ethmoidal osteoma. All of these tumors were attacked by exposure through an osteoplastic flap.

SUMMARY of two cases of diffuse osteoma of frontal bone and subjacent anterior fossa:

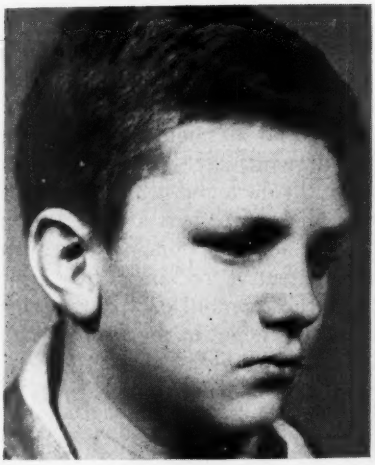
CASE 1. L. L., a white boy, aged 10, was admitted to the Medical College of Virginia Hospital on Jan. 14, 1949. Two years before admission, a gradual firm enlargement of the left frontal bone and supraorbital ridge was noticed. Its growth seemed to be accelerated following a blow on the forehead sustained five months before admission.

There were no subjective complaints, such as headache, vertigo, visual failure or diplopia.

Examination disclosed a non-tender, bony hard, diffuse swelling in the left frontal bone, involving also the left supraorbital ridge (fig. 1). Examination of the eyes by Dr. DuPont Guerry disclosed no loss in visual acuity, no visual field defects and no abnormality in the optic fundi. X-ray examination of the skull showed a dense, white, diffuse thickening of the left frontal bone which involved the roof of the left orbit (fig. 2), the anterior fossa on the left side as far posteriorly as the sphenoidal ridge. A preoperative diagnosis of diffuse osteoma was made involving the left frontal bone, orbit and left anterior fossa.

At operation (January 19) under satisfactory endotracheal (ether) anesthesia, the eyelids were first sutured with interrupted silk mattress sutures;

a left-sided frontal scalp flap of generous proportions was turned down. On the surface of the exposed skull, tumor presented as a rounded eminence, slightly more vascular than normal bone, occupying a position directly above



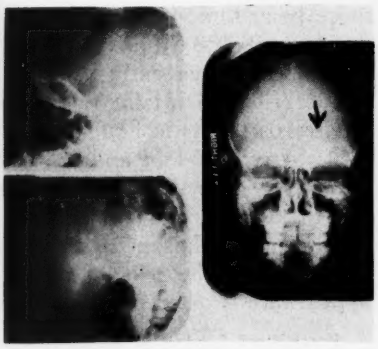


Fig. 1 Fig. 2

Fig. 1. (Case 1.) Preoperative photograph showing rather marked bony swelling in left supraorbital region and slight depression of left eye.

Fig. 2. (Case 1.) Preoperative skull films showing faintly outlined bony density of tumor in left frontal region (below arrow) and marked depression of left supraorbital ridge.

the left supraorbital ridge. Several trephine openings were made around the mass and connected with rongeurs. The tumor, when eventually removed, was seen to involve the entire left supraorbital ridge, the pterion, the roof of the left orbit, the anterior fossa back as far as the sphenoidal ridge, and medially it involved the ethmoid bone. The thickest portion of the tumor in the anterior aspect of the orbital roof and the floor of the anterior fossa was slightly over 3 cm. There was no definite line of demarcation from normal bone. It was spongy and friable but only moderately vascular. It was more reddish in appearance than normal bone and considerably more cancellous. In the course of removal, the ethmoid bone was largely removed and the air cells entered. Gelfoam was packed over the exposed air sinuses and into the region between the bulging capsule of the orbit and the frontal lobe. The entire procedure was carried out extradurally. One hundred thousand units of penicillin solution (5,000 u/c.c.) was instilled into the extradural space and the scalp closed with interrupted silk sutures without drainage, a pressure dressing being applied to the left eye. The microscopic diagnosis of the removed tissue was "benign osteoma."

Postoperatively the child made an uneventful recovery except for a transient diplopia. The preoperative prominent left supraorbital region was now concave (fig. 3). Three and a half months later (May, 1949), x-ray films of the skull having disclosed no remaining osteoma or infection, a tantalum plate

repair of the skull defect was carried out which restored satisfactorily the contour of the skull.

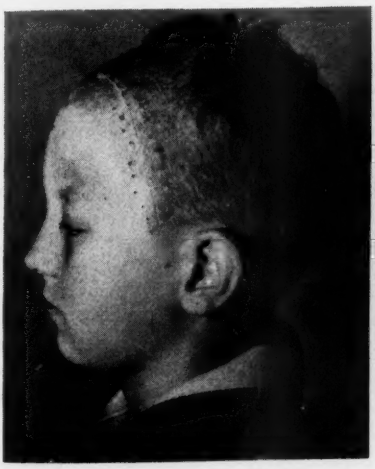


Fig. 3

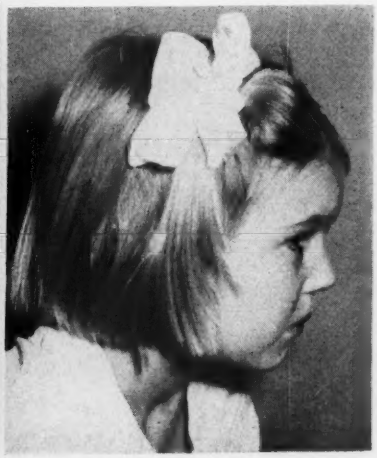


Fig. 4

Fig. 3. (Case 1.) Postoperative photograph showing left inferior frontal concavity, site of previous marked bony prominence (cf fig. 1).

Fig. 4. (Case 2.) Preoperative photograph showing diffuse bony prominence in right frontal region extending to or beyond midline. The protuberance was even more marked than the illustration suggests.

CASE 2. V. S., a white girl, aged 9, was admitted to the Medical College of Virginia Hospital on Jan. 17, 1949, during practically the same hospital period as the case just reported. Five years previously, at the age of 4, diffuse swelling in the frontal bone was first noticed. One year before admission she had struck her forehead on a fence which seemed to increase the rapidity of growth. Following the blow, the right eye slowly became more prominent. One month before admission, aching pain was first observed by the patient in the frontal region.

On examination, there was moderate protuberance noticed in the right frontal region extending to or beyond the midline (fig. 4). The right eye was slightly depressed. The prominent area of the skull was not tender to palpation. The right supraorbital ridge was lower than the left. The optic discs were well outlined, visual acuity and visual fields were normal. X-ray films of the skull showed thickening of the frontal bone, especially on the right side and of the right wing of the sphenoid bone. The diffuse thickening extended into the right ethmoid cells, which were grossly involved in the process. The preoperative diagnosis was probable diffuse osteoma, with osteosarcoma and meningioma also considered as possibilities.

A two-stage attack on the tumor was carried out by the resident, Dr. James Walker (January 22 and 28), the patient being under endotracheal (ether) anesthesia. Essentially the same operative approach was carried out as in

Case 1 (supra), a coronal scalp flap being used, however, as the tumor extended to each side of the midline (fig. 6). It was necessary to remove the lateral half of the right sphenoidal ridge and a portion of the greater wing

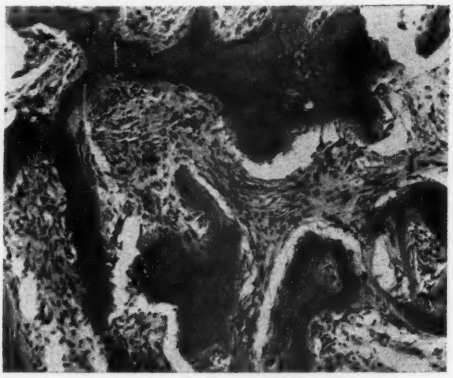


Fig. 5

Fig. 5. (Case 2.) Microphotograph of tumor, typical of benign osteoma. Section shows bony trabeculae separated by adult spindle cell fibroblasts. Diagnosis: Benign osteoma. Hematoxylin and eosin x 100.

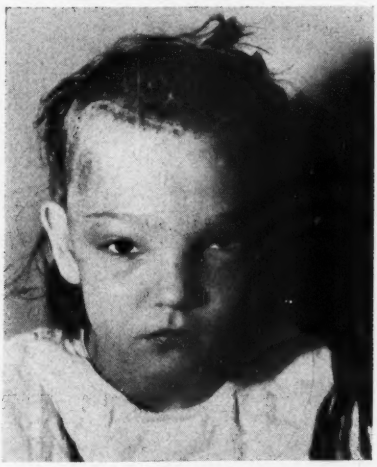


Fig. 6

Fig. 6. (Case 2.) Postoperative photograph after second-stage procedure at which time a tantalum plate was also inserted over the bony defect. The prominent frontal protuberance has been eliminated and the preoperative depression of the right eye and supraorbital ridge has disappeared.

of the sphenoid. The poles of both frontal lobes were exposed extradurally in the course of removal of the tumor. At the conclusion of the second stage, a tantalum plate was inserted. The total weight of the removed tumor was 85 Gm. The microscopic diagnosis was "benign osteoma" (fig. 5).

The postoperative course was uneventful and the patient was discharged from the hospital on Feb. 8, 1949, eleven days after the second-stage operation. The prominent right frontal region and preoperative depression of the right supraorbital ridge had disappeared (fig. 6).

Discussion of Cases 1 and 2: These two cases represent diffuse osteomata of the frontal bone involving the subjacent structures: roof of orbits, optic foramina, sphenoid wing, anterior clinoids, frontal and ethmoidal sinuses. The extent of the basilar removal calls for nice surgical judgment, as such tumors approach, if not actually impinge on, important structures such as the internal carotid artery and optic nerves, as well as the 3rd, 4th and 6th cranial nerves. Fortunately, the frontal sinuses were just beginning to develop in these two patients; normally, the frontal sinus doesn't reach even the level of the roof of the orbit until the eighth year of life;[4] they are rarely visible on x-ray examination until the sixth year of life, when they extend into the base of the vertical plate of the frontal bone. The ethmoids, however, are definitely present during the first, second or third years of life. These bony tumors grow very slowly and it is doubtless the wiser procedure occasionally to leave a small portion of the medial basilar portion of the tumor rather than attempt a too heroic removal of every vestige of the basilar growth because of the probability of irreparable damage to vital or important structures. On the other hand, the possibility of osteosarcoma developing (in remnants of the tumor left in situ) at a later date must be considered.

These cases are not to be confused with the pedunculated orbitoethmoidal osteomata as described so graphically by Cushing in 1927, which, although they also produce prominence and proptosis of an eye, are essentially discrete bony masses, arising as ethmoid "stones" in those sinuses, and *secondarily* involving the orbit and the supraadjacent frontal bone. They may then traverse the dura and impinge directly on the frontal lobe. They are best removed through a frontal craniotomy. The only feasible method for removal of the type of case just described in this paper is anterior *craniectomy* or piecemeal removal of the greatly thickened bone with rongeurs to the extent of the tumor in the vault and base.

The following case is of a different order of osteoma from the two previous examples, both in location and type of growth. It is essentially an osteoma arising presumably from the inner table of the temporal bone, discrete in character, eroding grossly through the dura, markedly indenting the underlying temporal lobe and producing a clear-cut contralateral homonymous hemianopsia. It was removed in toto, followed immediately by a tantalum plate insertion, with postoperative recovery.

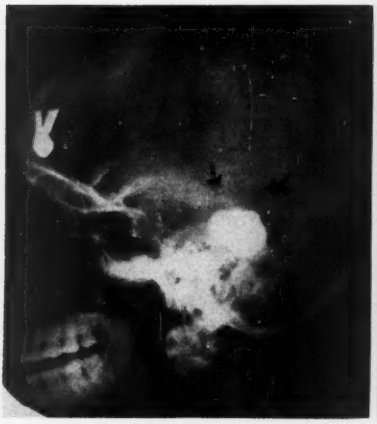


Fig. 7

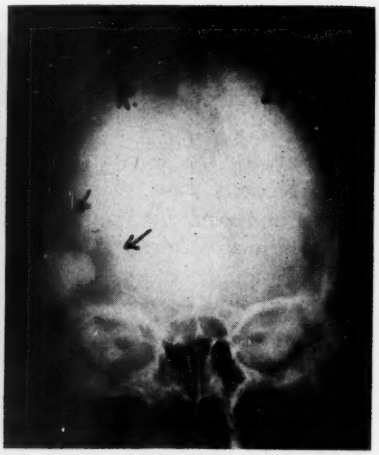


Fig. 8

Fig. 7. (Case 3.) Preoperative right lateral skull film. The discrete bony tumor is clearly outlined (arrows) and measured 3 by 3 cm. in transverse diameters. Its close approximation to the mastoid cells and sigmoid sinus is apparent.

Fig. 8. (Case 3.) Preoperative anteroposterior skull film. The cone-shaped formation of the osteoma is clearly shown (arrows). The tumor penetrated the dura and grossly indented the underlying temporal lobe producing a clear-cut contralateral homonymous hemianopsia.

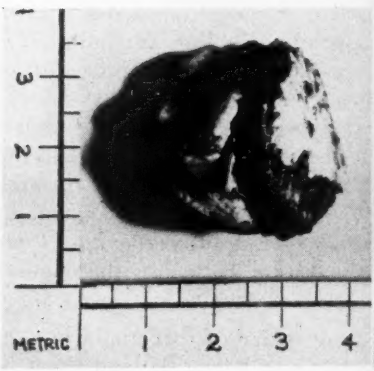


Fig. 9

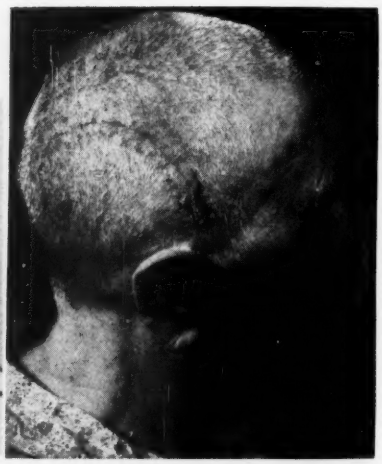


Fig. 10

Fig. 9. (Case 3.) Photograph of the osteoma removed en bloc. Remnants of dura are apparent along vertical equator of the tumor. All the growth to the left of this line was intradural producing a gross cone-shaped concavity of the underlying temporal lobe. Weight 18 Gm.

Fig. 10. (Case 3.) Postoperative photograph showing line of incision and well-healed scalp flap beneath which is a tantalum plate overlying the fibrin film used to cover the dural defect.

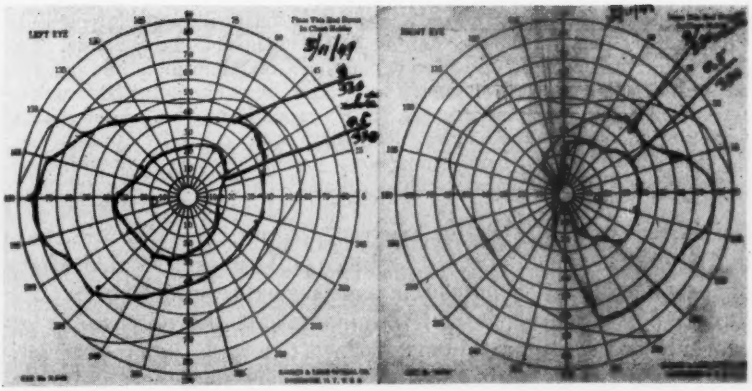


Fig. 11. (Case 3.) Postoperative visual fields (eleventh postoperative day). Considerable improvement is noted (especially in left eye but also beginning to appear in the right) over the preoperative fields which had shown a complete left homonymous hemaniopsia with vertical bisection of the macula (see text).

Case 3. Mrs. R. C., a white woman, aged 69, was admitted to the Medical College of Virginia Hospital on April 27, 1949, complaining of right-sided headache and inability to see to the left of the midline. The headache had been present for three years. There was no history of trauma. She was also a hypertensive and a mild diabetic. The past history included bilateral retinal hemorrhages, probably of diabetic or hypertensive origin. Neurologic examination disclosed no objective abnormality except a definite complete left homonymous hemianopsia with bisection of the macula. X-ray examination of the skull showed, just above the right mastoid, an area of greatly increased density 3 by 3 cm. in diameter which appeared completely calcified. The long axis of the tumor was pointing inward from the inner table of the temporal bone; the base was considerably wider than the tip, giving the growth a cone-shaped appearance pointing inward (figs. 7 and 8). There was no palpable lesion externally on the patient's head. The preoperative diagnosis was osteoma indenting the right temporal lobe causing a contralateral homonymous hemianopsia.

At operation (April 30), under endotracheal (ether) anesthesia, a small osteoplastic flap was first planned in the right temporal region (surrounding the osteoma); the trephine openings and the connecting saw-cuts were made. In attempting to elevate the bone flap, however, it was discovered that the growth had traversed the dura widely and was rather firmly imbedded in the underlying temporal lobe. The bone flap was then rongeured away until the edges of the bony tumor were approached, following which the dura was incised around the growth and the involved skull, dura and discrete bony tumor itself removed en bloc (fig. 9), precisely as one would remove a meningioma of the cerebral convexity that had eroded through the dura and widely and firmly involved the overlying skull. The incision in the dura was very close to the right lateral and especially the sigmoid sinus extending to within 1 to 2 mm. of these structures (fig. 7); also, the mastoid cells were entered of necessity in the preliminary mobilization of the tumor and the involved skull. The opened mastoid cells were packed immediately with Gelfoam. The tumor,

a typical stony hard, discrete osteoma, had deeply indented the underlying right temporal lobe, thus accounting for the left homonymous hemianopsia. The dural defect was covered with fibrin film and a tantalum plate inserted over the skull defect at the same session. The removed tumor weighed 18 Gm. The patient made an uneventful recovery, the operative wound healed per primam (fig. 10), and she left the hospital eleven days postoperatively. At that time, the clear-cut left homonymous hemianopsia, present preoperatively with bisection of the macula, had widened out considerably (fig. 11).

Discussion of Case 3: Pilcher[5] reported an almost identical case in 1946 which he designated "osteoma of the inner table of the temporal bone," although pathologists would perhaps prefer the term endostosis, basing their terminology on the microscopic appearance of the tumor. He emphasized that such tumors may seriously compress the brain resulting in pressure symptoms and sometimes in epilepsy. The pathologic designation of ossifying meningioma would also be acceptable in Case 3.

Courville and Crockett,[6] in 1948, reported an interesting case of a hyperostosing osteoma involving both parieto-occipital areas across the midline. The x-ray appearance preoperatively strongly suggested hyperostosis over a meningioma because of its typical "sun-ray" appearance. It was removed en bloc, the dura opened bilaterally to either side of the longitudinal sinus and no meningioma or other tumor found. Microscopic examination of the removed extradural bony mass disclosed it to be a typical benign solid or eburnated type of osteoma with radiating spicules in the outer portion of the tumor.

Rand,[7] in 1922, removed a lesion similar to that in Case 3 in the right frontal region. His patient had convulsions and left-sided weakness. The bony tumor in Case 3, involving, as it did, the right temporal bone and a small portion of the middle fossa, was an entirely discrete lesion, had widely traversed the dura and markedly depressed the underlying right temporal lobe producing a left homonymous hemianopsia. The first two (frontal) cases were, on the other hand, *diffuse* osteomata with no line of demarcation, involving the extradural structures only, including the orbits, frontoethmoid region, and the anterior fossa as far back as the sphenoid ridge; they require piecemeal removal with rongeurs. The propriety of inserting immediately tantalum plates in Cases 2 and 3, in which the ethmoid and mastoid cells were entered, respectively, may be questioned but fortunately no untoward effect resulted from such a procedure in these two cases.

Summary

Three cases of benign osteoma of the cranial vault and base are

presented, illustrating the surgical problems of two varieties of this type of tumor.

1. Two of the cases (1 and 2) were diffuse osteomata of the frontal bone in children aged 9 and 10, producing great thickening of the vault *and* anterior fossa and intermediate structures, treated by piecemeal removal (partial craniectomy) extradurally, until normal bone was encountered.

2. The final case (3) was a discrete osteoma of the right temporal bone in an elderly patient (aged 69) grossly eroding the dura and markedly indenting the underlying right temporal lobe, with the production of a contralateral homonymous hemianopsia, which visual defect improved postoperatively.

All three postoperative bony defects were covered with a tantalum plate either at the time of removal of the tumor or at a later session. Uneventful recovery ensued in each instance.

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EWING'S TUMOR OF THE VERTEBRAE*

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SOMEWHERE about the time the Bone Registry was organized, in 1921, Ewing[1-3] published his observations on the tumors which he called "Endothelial Myeloma," and which the literature, owing to the controversy regarding its origin, has since designated as "Ewing's tumor."

Owing to the efforts of the Bone Registry and to the pooling of cases, a great deal has been learned about the course, incidence, and response of this tumor to different therapeutic measures, although its nature is still disputed.

Because of the uncommon occurrence of this tumor in the spinal column (about 4.7 per cent), we are presenting a case which had an interesting course.

Ewing[1-3] believed the tumor was derived from the angioendothelium. He described a cell type with a small polyhedral cell, with pale cytoplasm, small hyperchromatic nucleus with ill defined cell outline. Sections showed sheets of cells, the healthiest of which were in close proximity to the blood vessels giving a perithelial picture, but no rosette patterns.

Since the time of Ewing's original work many authors have taken issue with his idea as to the histogenesis of the tumor.

Colville and Willis[4] and Willis,[5] Geschickter and Rex[6] advanced the idea that the bone lesion was only a rapidly growing metastatic lesion of a neuroblastoma or a sympathoblastoma, and they stressed the fact that many reported cases of Ewing's tumor had very scanty autopsy reports, or were biopsy diagnoses, and might have been metastatic neuroblastoma or sympathoblastoma. This view was also expressed by Tilestone and Wolbach.[7]

Connor,[8] McJunkin,[9] Sabin,[10] Maximow,[11] and Oberling[12] all stress and support the theory of a reticulo-endothelial origin of Ewing's tumor with Oberling[12] recognizing three forms of reticulo-endothelial tumors. Lichtenstein and Jaffe[13] also support the reticular origin of the tumor.

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Hamilton[14] states that Ewing's tumor arises from the blood vessels or perivascular lymphatics.

Hirsch and Ryerson[15] cite the similarity of Ewing's tumor to the bone metastases of carcinoma of the lung.

Melnick[16] in his article states that tumors may be classified as of endothelial origin if they have or exhibit (1) demonstrable origin from endothelium, (2) vasoformative properties, (3) cell types resembling the endothelial cells, or (4) perivascular or alveolar arrangement of the cells. He feels that in only the first two instances is one on safe ground in classifying the tumors as of endothelial origin. Melnick[16] believes Ewing's tumor to be an undifferentiated round cell sarcoma, probably originating from embryonic mesenchymal cells in the connective tissue about the blood vessels in the Haversian canals. Neely and Rogers[17] agree with Melnick[16] as to the histogenesis of the tumor. Kolodny[18] states that "there is no sufficient proof on hand to support the contention of Ewing that "diffuse endothelioma" (Ewing's tumor) "is really endothelioma." It is obvious from the foregoing that the histogenesis of the tumor is unsettled and that the debate is heated.

Incidence

Meyerding and Valls[19] in a review of 424 cases of bone tumor found 114 cases of Ewing's tumor or approximately 27 per cent, Heublein, Moolten and Bell[20] state that "Ewing's tumor constituted 42 per cent of all bone tumors seen at Percy Jones General Hospital."

In a review of the literature, we have gathered 318 unselected cases of Ewing's tumor which definitely mention the bone involved. This review probably leaves many cases unaccounted for. The incidence of the various bones involved is illustrated in Table 1.

In the cases reviewed, 3 cases of Ewing's tumor involving the vertebrae showed some characteristics similar to those in the case we are reporting, i.e.: paralysis, hypoesthesia, urinary retention or incontinence, bowel incontinence, hyperactive reflexes, soft tissue involvement by the tumor and marked improvement following roentgen therapy.

One case reported by Ewing[2] showed a paraplegia, this patient was treated with roentgen therapy and was free of symptoms for three years. The vertebrae involved and the amount and type of therapy given were not discussed.

A second case reported by Cabot[21] showed paraplegia, paresthesia, hematuria, hematemesis and respiratory difficulty. This tumor

involved the fourth dorsal vertebra, and unfortunately the patient died soon after biopsy and before therapy.

Pomeranz[23] reported a case involving the arches, body and transverse processes of the third lumbar vertebra, with extension of the tumor mass into the surrounding soft tissue. This case had paralysis of both lower limbs and bladder, and bowel incontinency. There was marked improvement following radium pack therapy consisting of: (1) 8,040 mg. hr. over 8 by 10 cm. field at a focal skin distance of 6 cm. with 2 mm. lead filter, delivered posteriorly, (2) 6,365 mg. hr. delivered anteriorly with the same factors as in No. 1, (3) repeat of 8,040 mg. hr. to the posterior field.

TABLE I

Bone Involved			*Incidence*	*Per Cent*
Femur			65	20.4
Tibia			42	13.2
Fibula			19	5.9
Radius			7	2.2
Ulna			9	2.8
Humerus			28	8.8
Phalanges			1	.3
Metacarpals			4	1.3
Bones of the foot	Calcaneous ..... 2 Tarsal ..... 1 Metatarsals .... 1 Others 8	Total	12	3.7
Pelvic bones	Pubis ..... 6 Ilium .....14 Ischium ..... 1 Others 12	Total	33	10.3
Vertebrae			15	4.7
Sternum			2	.6
Ribs			19	5.9
Clavicle			12	3.7
Scapula			15	4.7
Mandible			8	2.7
Sacrum			10	3.1
Maxillae			7	2.2
Skull	Temporal bone.. 1 Mastoid bone... 1 Parietal bone.... 2 Others 6	Total	10	3.1

Ewing's tumor of the vertebrae appeared 15 times in 318 cases reviewed, an incidence of 4.7 per cent.

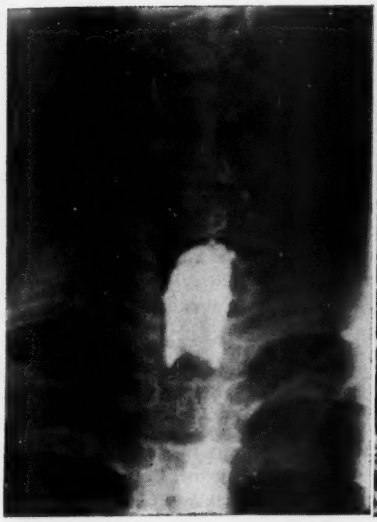

Fig. 1

Fig. 2

Fig. 1. Lipiodol studies of the descending type showed a complete block at the upper border of the third dorsal vertebra with the concavity of the lipiodol column, suggesting an extensive extradural growth associated with a primary bone tumor (hemangioma).

Fig. 2. Microscopic examination of the tumor tissue revealed cells of uniform size with indistinct cell outlines, scanty cytoplasm and small pale vesicular nuclei which were rounded or oval. The growth was very vascular and foci of hemorrhage and necrosis were frequent.

Case Report

M. W., a 17 year old white female, was admitted to the Medical College of Virginia Hospital with the chief complaint of severe pain between the shoulders, and weakness of the lower extremities. She gave a history of recurrent pains between the shoulders for the past four years. Three weeks before admission the pain became continuous and was intensified by coughing and sneezing. One week prior to admission she developed marked weakness of both legs with numbness which extended upward to a level two inches below the nipple line.

Physical examination showed a spastic paraplegic with hypesthesia extending from about two inches below the nipple line downward and including the legs. Reflexes were hyperactive in both legs. There was also urinary retention.

Lumbar puncture revealed a slight increase in the spinal fluid pressure, which soon dropped to normal following the removal of a small amount of fluid. The Queckenstadt test was positive and pressure over the abdomen gave an increase in the flow of spinal fluid.

Laboratory findings were as follows: red blood cells, 4,280,000; hemoglobin, 84 per cent; white blood cells, 8,300; lymphocytes, 15 per cent; large monocytes, 3 per cent; polymorphonuclears, 60 per cent; eosinophils, 2 per cent; and basophils, 1 per cent. The urine had a specific gravity of 1.025, and a

1 plus albumin. The spinal fluid showed a total cell count of 6 cells, polymorphonuclears 5, and 1 lymphocyte. The Wassermann test was negative.

Roentgen findings revealed a rarefied trabeculated appearance of the third dorsal vertebra without compression, and with the intervertebral spaces preserved. Lipiodol studies of the descending type showed a complete block at the upper border of the third dorsal vertebra with the concavity of the lipiodol column, suggesting an extensive extradural growth associated with a primary bone tumor (hemangioma) (fig. 1).

At operation a grayish-pink, meaty, soft, vascular tumor was encountered just under the fascia of the erector spinae group of muscles. The tumor was invading the muscle tissue and appeared to be very extensive. Frozen sections revealed a sarcoma, most probably Ewing's tumor.

Microscopic examination of the tumor tissue (fig. 2) revealed cells of uniform size with indistinct cell outlines, scanty cytoplasm, and small pale vesicular nuclei which were rounded or oval. The growth was very vascular and foci of hemorrhage and necrosis were frequent.

The patient was given roentgen therapy consisting of a total tumor dose of 1600 r. Following therapy she showed marked improvement, being able to move her legs and void. Six weeks later she was given additional roentgen therapy to a total tumor dose of 1,148 r. The patient was then lost to follow-up until her death 25½ months later.

Treatment

There are numerous five year survivals of Ewing's tumor reported in the literature.

The treatment has run the scale of surgery, toxins, and irradiation or their various combinations. In reviewing the literature it appears that the various methods used have given comparable results.

In the treatment of bone tumors Ewing's tumor would seem to be the choice for irradiation therapy because as pointed out by Herendeen[23] it is the bone tumor most sensitive to irradiation. However, success with radiation appears to fall somewhat short of the results obtained by irradiation followed by surgery where possible (Kahn[24]).

Regardless of the form of therapy chosen the prognosis is grave. This is especially true of those patients who present themselves with fever, leukocytosis and an increased sedimentation rate. These patients have a shorter survival time than do those who present themselves without the above findings. These facts were pointed out by Lichtenstein and Jaffe.[13]

If irradiation is given, we should not treat so heavily that the healthy tissue is destroyed or the natural defenses broken down (Herendeen[23]).

Pfahler[25] states that "if radiation alone is used, we should expect good local response but ultimate failure."

Summary

1. The histogenesis of Ewing's tumor is still disputed and a brief review of the various ideas has been presented.

2. In a review of 318 unselected cases of Ewing's tumors the vertebrae, exclusive of the sacrum, were involved fifteen times, or 4.7 per cent.

3. In the above fifteen cases, three of which had a complete history and physical examination, symptoms of spinal cord involvement or pressure were present. These, together with our own case, make a total of four cases with spinal cord involvement or pressure, three of which showed marked improvement following irradiation therapy.

4. The case presented survived 73½ months following the onset of symptoms, and 25½ months following admission.

5. Roentgen therapy is in most instances only palliative, but there are some long term survivals known to exist.

6. The present trend in therapy appears to be irradiation followed by radical surgery.

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HEMANGIOENDOTHELIOMA: REVIEW OF THE LITERATURE WITH A REPORT OF TWO CASES*

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GOLGI[1] introduced the term endothelioma in 1869, and Kolaczek[1] in 1895 used the term to denote tumors with blood between the acini. Even in the first decade of the twentieth century the term was applied to all tumors which could not be satisfactorily diagnosed. This led to the inconsistency of the histologic pattern, and clinical course of the cases described in the literature as endothelioma. However, with the stabilization of what should be considered as endothelium, namely, the single layer of squamous cells which lines the inner surfaces of blood and lymph vessels and sinuses, as well as the heart, the term endothelioma became fixed to designate malignant new growths which arise from the above mentioned structures.[2]

Ribbert[1] categorically stated that it had not been shown conclusively that a neoplasm arose from endothelium, and MacCallum believed few endothelial tumors had been described. Borst[1] and Ewing accepted the broader application of endothelial origin of the tumor.

Vascular tumors present a wide histogenetic range which in order of activity may be grouped as follows:

a. The typically benign angioma,

b. The multicentric and benign angioma,

c. The cellular capillary angioma which occurs in children and is locally invasive (Hemangioma Hypertrophicum),

d. The locally invasive, plexiform angioma which may invade muscle and even bone,

e. The genuine malignant angioma or true hemangioendothelioma. Considerable controversy appears in the earlier literature concerning so-called metastasizing benign angioma as reported by Borrmann,[1] Rabson,[3] and Klinge[4] under various titles. This group of angiomas appears histologically benign, but clinically behaves as malignant growths, e.g. local invasion and destruction, recurrence following excision, and so-called metastases to distant organs. Jaffe,[5] on the contrary, believes these cases are multiple tumors and not true metastases, but arise coincidentally from multiple congenital

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foci. This would be in keeping with the theory of local origin of vascular tumors. It is now well accepted that malignant angiomas exist. In their histogenesis these tumors show a tendency to revert to the original primitive mesenchymal tissue, giving rise to a varied histologic picture. One essential feature as emphasized by Thomas[4] is the "angioblastic tendency as evidenced by vasoformative process of endothelial proliferation and formation of new blood vessels."

Numerous classifications have been advanced, all having certain merits. Pulford[6] classified endothelial tumors on biologic basis into (1) Angioma, (2) Angioendothelioma, and (3) Endothelioma. The classification in Thomas's paper was:

(1) Angioma, benign
(2) Angioma, malignant
    (a) Angioendothelioma
    (b) Angiosarcoma

Considerable confusion exists over use of the terms angioendothelioma and angiosarcoma. Winkler considers endothelioma, periendothelioma, angiosarcoma, and sarcoma of vascular tissue as synonymous terms. Ewing,[1] on the other hand, suggested angiosarcoma be limited to cellular angiomas in which the unit is the vessel and not the endothelial cell, and angioendothelioma be reserved for vascular tumors in which the endothelial cell predominates.

The tumor may occur at any age and shows little sex predilection. Malignant angioma of bone, however, appears to be more common in the second and third decades.[7] The larger series that have been reported show that the neoplasm has occurred more commonly in the skin, subcutaneous tissue, and skin appendages. The other organs are about equally involved.

Metastases occur by way of the blood stream as a rule, but occasionally by the lymphatics. The lungs are most frequently involved with metastases to liver, spleen, kidneys, pancreas, adrenals, bones, skin, lymph nodes, and peritoneum reported.

The pathologic picture varies, but in the main consists of wide sheets of cells with large pale oval nuclei, with a moderate amount of clear staining cytoplasm and well defined cell borders. Few mitotic figures are the rule. These atypical hyperchromatic endothelial cells tend to form vascular channels, some of which contain red blood cells. Stout[8] outlines the criteria of formation of atypical endothelial cells in greater numbers than are required to line the vessels with a simple endothelial membrane, the formation of vascular tubes with a delicate framework of reticulin fibers, and a marked tendency for their lumens to anastomose as the striking

features of growth of the tumor. Silver connective tissue stains are often helpful.

A clinical diagnosis is practically impossible and definite diagnosis rests on microscopic examination. Skeletal hemangioendothelioma, according to Constant,[9] must be considered in the differential diagnosis when the lesion appears by roentgenograms to be cystic, trabeculated, or invading soft parts.

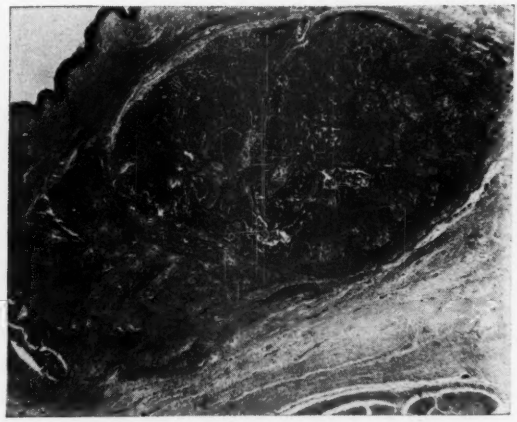
Hemangioendothelioma is listed at the bottom of the radiosensitive tumors by Desjardins. Baumann and Schenke[10] believe its radiosensitivity approaches that of lymphoblastoma. Janes and Ghormley[11] of the Mayo Clinic reported two cases treated with radioactive phosphorus and deep roentgen therapy and suggested that it possibly prolonged the period between exacerabations, but Popp, of the Section on Roentgen Therapy at the Mayo Clinic, was of the opinion that such results could reasonably be expected from roentgen rays alone.

Early diagnosis and radical surgical treatment are essential for cure. Postoperative irradiation should be carried out in protracted form to a tumor dose of 3000 r when complete surgical extirpation is in doubt.

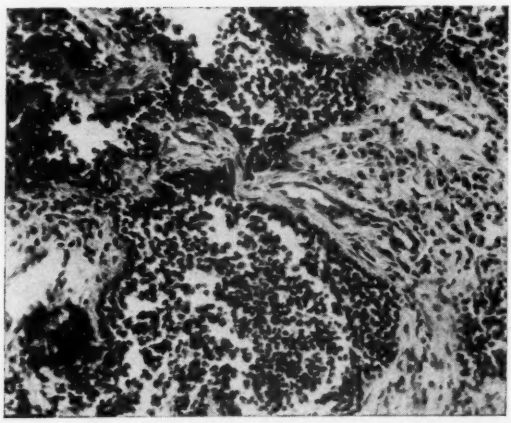
CASE 1. S. H., a 5 year old white female, was admitted to the Medical College of Virginia Hospital, for the first time, on Sept. 30, 1948, with a chief complaint of "swelling in the neck." The child had been well until March, 1948, at which time she had had what her mother thought was acute tonsillitis. The patient vomited a half cupful of material that was the color of "rotten eggs," and she had an elevation of her temperature for a few hours. Three days later a knot the size of a walnut appeared, and at this time she was running a low grade fever. She was treated by several physicians, one of whom gave her penicillin which had no apparent effect. She also had two minor operations which were thought to be biopsies, and the mother was told that the child had cancer. She consulted still another physician, who in turn referred her to our hospital.

On admission her temperature was 99.8° F., pulse 100, respirations 24, and blood pressure 100/60. She was a well developed, well nourished, white child in no acute distress. There was a hard mass on the lower aspect of the body of the right mandible measuring 5 to 6 centimeters in diameter. The mass was non-tender and in a small part of the mass there was a bluish discoloration of the skin. The mass was fixed, and there was no fluctuation. The mass did not protrude into the oral cavity. Three centimeters below the angle of the jaw there was another mass, 2 cm. by 2 cm., which was freely movable, and thought to be a lymph node. Several lymph nodes were noted in the posterior triangle of the neck. There were small nodes in the axillary and inguinal regions. The physical examination was otherwise normal. Laboratory examinations showed a red blood count of 5,200,000 cells per cu. mm., Hemoglobin 15.8 Gm., white blood count 5,200 cells per cu. mm., with 30 per cent polymorphonuclear neutrophils and 69 per cent lymphocytes. The urine was negative, and the flocculation test was negative. X-ray examination of the

mandible showed a soft tissue density 3.5 cm. by 6.5 cm. by 4.0 cm. lateral to and slightly below the body of the right mandible with periosteal elevation for 1.6 cm. and with no bony destruction. The findings were suggestive of osteomyelitis.



S49-1003X60 Case 1



S49-1003X400 Case 1

Case 1: S49-1003 X 60—Shows an irregular tumor nest in the subcutaneous tissues with irregular blood channels lined by sheets of tumor cells. S49-1003 X 400—Shows the way the sheets of endothelial cells merge into the discrete cells in the lumen of the sinus.

A review of the biopsy specimen taken elsewhere was reported as follows: "The section displays a tumor composed chiefly of rather small cells with round and oval vesicular nuclei which for the most part are rather uniform. Some nuclei, however, are larger than others and an occasional mitotic figure is seen. In some sectors the cells are arranged in strands and sheets while in

others there is definite tendency to form vascular channels. Occasional vascular channels are engorged with blood, but for the most part only a moderate number, and occasionally only scattered erythrocytes are present among the neoplastic cells. There is a network of fibrocollagenous tissue, the trabecullae of which often support normal appearing vessels. We regard the prominent cells as endothelial cells, and feel that the neoplasm is of vascular origin. We believe it should be classified as a hemangioendothelioma." Pathologic diagnosis: "Hemangioendothelioma, right side of neck."

On October 8 x-ray therapy was begun. Factors were 130 Kv, filter ¼ Cu, ½ mm. Al, distance 23 cm. Daily for six days 200 r was given to a right anterolateral portal (dosage measured in air), and then 200 r (measured in air) daily for four days to a right posterolateral portal. Total dosage was 2,000 r measured in air. Following the x-ray therapy there was marked regression in the size of the tumor, and the patient was discharged on October 27, to be followed in our Tumor Clinic.

The patient was readmitted to the hospital on Nov. 30, 1948, at the suggestion of the tumor board, for radical excision of the tumor overlying the right mandible. The mass had receded considerably, almost disappearing, but in recent days had seemed to enlarge. On December 9, the patient underwent an operation. The tumor was removed by block dissection, including most of the myohyoid muscle, the periosteum of the lower aspect of the mandible, most of the submaxillary gland, which was within the tumor, and the submaxillary lymph nodes. The gross pathologic report was as follows: "The specimen consists of a mass of pinkish tissue of firm consistency measuring approximately 6 cm. in diameter. On section the tumor appears to be composed of firm yellowish-white tissue." The microscopic description was as follows: "Sections show tumor which has infiltrated skeletal muscle, connective tissue, fatty tissue, and salivary glands. The periosteum is not involved. In certain areas the tumor cells surround and line tortuous spaces, in others, more or less solid sheets are observed. The tumor cells are polyhedral with ill-defined cell outlines. The cytoplasm is granular. The nuclei are uniform, rounded and oval. The nuclear membranes are delicate. The chromatin is arranged in irregular granules and the nucleoli are as a rule multiple and small. Mitotic figures are scarce. Scattered throughout the tumor especially towards the periphery are cells with dark pyknotic nuclei (radiation effect). Where the tumor is in the form of sheets, Masson stain shows beginning collagen deposition. Both Masson and silver stains show the absence of any fibrillary structures related to the tumor cells. Heidenhains iron hematoxylin shows the tumor cells to have granular cytoplasm. No striations of any type are found.

"In some fields the tumor cells are traceable to the endothelial lining of the capillaries in the section. Pathologic diagnosis—Hemangioendothelioma invading skeletal muscle, connective tissue, and adipose tissue and salivary gland."

The operative wound healed well and the patient was discharged on Dec 29, 1948, to be again followed in the Tumor Clinic.

Because of the development of enlarged submental glands, and a nodular mass at the anterior border of the right sternocleidomastoid muscle, she was readmitted to the hospital on Jan. 28, 1949. A radical neck dissection was advised by the tumor board. This was performed on January 29. The pathologic report was as follows:

"Tissue composed of—

1. striated muscle

2. salivary gland
3. lymph nodes
4. tumor

There is no tumor infiltration in any part of the first three structures. The structure of the tumor does not differ from the description of S-48-5516 (the previously reported specimen). Few mitotic figures were noted. Pathologic diagnosis—Hemangioendothelioma."

The postoperative course was uneventful and the patient was discharged on February 8, to be followed in the Tumor Clinic.

The patient was readmitted to the hospital on March 1, 1949, because a nodule was noted in the old operative scar. This was excised and the pathologic report was hemangioendothelioma infiltrating striated muscle and subcutaneous tissue. X-ray therapy was again started, and 3,000 r measured in air was delivered to each of two portals, left lateral and right lateral neck, in twenty days. Factors were 130Kv, filter ¼ Cu, ½ Al, distance 20 cm., size of portals 10 x 8 cm. Total dosage 6,000 r measured in air.

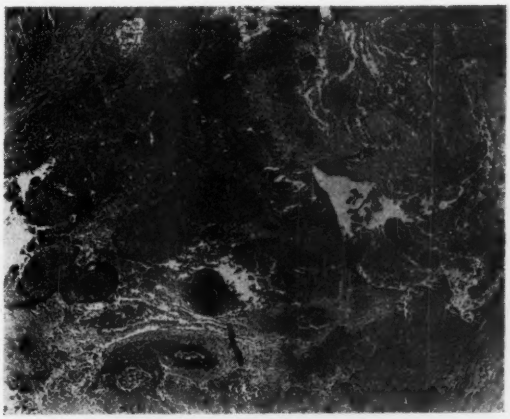
On May 6, 1949, she was again admitted to the hospital, her fifth admission, because of severe flank pain. An intravenous pyelogram was interpreted as showing normal findings, but films of the dorsal spine showed compression fractures of the 8th and 10th dorsal vertebrae. The x-ray examination of the chest was negative for metastatic lesions, and films of the skull showed no metastases.

The patient's temperature remained elevated, staying between 102° F. and 106° F. and clinically she progressively got worse. She received blood transfusions, vitamins, and aminopterin. In addition she was given 1200 r to the dorsolumbar vertebrae by posterior portal, HVL 0.9 Cu. She steadily went downhill and died July 4, 1949. Permission for autopsy could not be obtained.

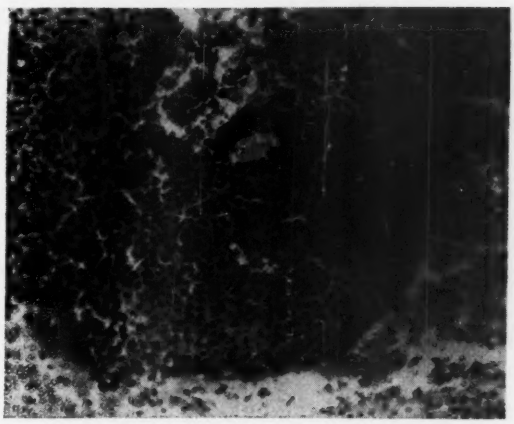
Case 2. J. E., a 26 year old colored male, was admitted to the Medical College of Virginia Hospital on Feb. 16, 1949, with a chief complaint of pain and swelling of the right ankle. One year prior to admission he had sprained his ankle while plowing. There was subsequent pain which subsided after one week. In September, 1948, he had injured his ankle again and developed a swelling which finally subsided. He went back to work, but continued to have pain in the ankle. On Jan. 26, 1949, he had consulted his local physician who took an x-ray of the right ankle, and on the basis of this film referred the patient to the hospital. On admission the patient was a well developed, well nourished colored male in no acute distress. There was a large, bumpy, fusiform swelling over the lower right tibia, rubbery in consistency, except for certain areas that were bony hard. No particular tenderness was noted. Large rubbery lymph nodes were noted in the popliteal and femoral regions. The physical examination was otherwise negative.

Laboratory examinations: red blood count 5,360,000 per cu. mm., hemoglobin 16.0 Gm., white blood count 5,200 per cu. mm. with 42 per cent polymorphonuclear neutrophils, 55 per cent lymphocytes, 2 per cent monocytes. The urine was negative. The flocculation was positive, the Wassermann negative. Calcium 9.8 mg. per cent, phosphates 3.4 mg. per cent. Alkaline phosphatase 1.8 B. U. A blood smear showed a few large reticuloendothelial cells. X-ray examination of the right ankle and lower leg showed bone destruction in the region of the medial malleolus, with periosteal elevation for about 12 cm. in the lower end of the tibia on both sides. There had been bone destruc-

tion 7 cm. from the distal end of the tibia in an area 5½ cm. x 2 cm. with possible attempted bone regeneration. There was periosteal elevation in the lower 11 cm. of the fibula with slight bone destruction anteriorly. Conclusion: Chronic osteomyelitis was favored over new growth. Film of the chest showed



S49-1003X60 Case 2



S49-892-400

Case 2: S49-892 X 60—The figure illustrates an irregular blood sinus with papillary projections of the tumor sheets into the sinuses with discrete tumor cells and blood cells in the sinus. S49-892 X 400—Shows a high power field of one of the papillary structures, the center of which is a vessel with the endothelial cells arranged in parithelial sheets.

no evidence of metastases or other pathologic changes. On February 17, a biopsy was taken and the pathologic report was as follows: "Microscopic description—1. Nodule from the tibia shows strands of collagenous connective tissue enclosing tortuous inter-communicating channels which are lined by

sheets of cells whose bodies are indistinct and the nuclei are rounded with granular chromatin, and multiple small nucleoli. Few mitotic figures are seen. 2. The lymph node shows metastases of the same tumor. Pathologic diagnosis—Hemangioendothelioma with metastases into the lymph nodes examined."

On February 22 a right mid-thigh amputation was performed, and the microscopic examination of the tumor showed a hemangioendothelioma invading bone, periosteum, and muscle. The postoperative course was uneventful and the patient was discharged March 5, 1949, to be followed in the Orthopedic Clinic. Reexamination on June 13, 1949, showed some enlarged right inguinal lymph nodes and he was referred to the hospital for a radical resection of this region. The dissection was carried out on June 14, and the microscopic report was as follows: "All specimens show different aspects of the same tumor as previous biopsy No. S-49-809. Pathologic diagnosis—Hemangioendothelioma." Roentgen therapy was advised and he received 1600 r (measured in air), HVL 0.45 Cu., to the right anterior inguinal region. A film of the chest showed no metastatic lesions. He was discharged from the hospital July 12, 1949. The patient was seen in the Tumor Clinic on Jan. 10, 1950, at which time he felt well. He had gained six pounds in weight since October, 1949. No nodes could be palpated in the right inguinal region and the liver was not palpable. An x-ray of the chest, however, showed a circumscribed area of increased density near the right lung base 3 cm. in diameter. This was interpreted as a metastatic nodule.

On Feb. 15, 1950, x-ray therapy to the lungs through four portals was started giving 250 r daily to each of two portals, 20 x 20 cm. left and right anterior chest, left and right posterior chest. The factors were 180 Kv, 20 MA, ½ cu., 1 Al. A tumor dose of 3,000 r was reached following which roentgenograms of the chest showed a decrease in the size of the metastatic nodule. The patient remains clinically well two years following the initial symptoms.

Summary

1. A review of the literature has been made and two case reports of hemangioendothelioma presented.

2. There is no clinical picture to identify this tumor, nor is the roentgenogram diagnostic. Positive diagnosis depends solely upon microscopic appearance.

3. Early diagnosis and radical surgical excision are paramount for cure.

4. Deep roentgen therapy is helpful when question of incomplete extirpation arises at time of surgery.

5. One of the patients is living two years after onset of symptoms, but has metastatic lesions in both lung fields. The other patient was dead sixteen months after onset of symptoms.

The authors wish to express their appreciation to Dr. Phillip Sahyoun of the department of Surgical Pathology for his help in reviewing the microscopic sections.

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A COMPARATIVE STUDY OF THE EFFECTS OF PARASYMPATHETICO-MIMETIC DRUGS ON THE URINARY BLADDER*

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PARASYMPATHETICO-MIMETIC drugs have been shown by various observers to cause contraction of the vesical detrusor muscle.[1] Among the more commonly used preparations available are acetyl beta methyl choline (Mecholyl), carbaminoyl choline (Doryl or Carbachol), and furfuryltrimethyl ammonium iodide (Furmethide). To our knowledge, no objective clinical comparison of the effects of these drugs on the urinary bladder has yet been reported. To determine whether or not there is a difference in the response of the vesical detrusor to these three drugs is the purpose of this study.

The pharmacology of parasympathetico-mimetic drugs is fairly well understood. They simulate the action of acetylcholine, the chemical mediator liberated at the myoneural junction on stimulation of parasympathetic nerves. Acetylcholine is not used clinically due to its rapid destruction by cholinesterase. Mecholyl and Doryl are also choline esters, but their duration of action is many hundred times that of acetylcholine due to slower destruction and excretion. Furmethide is a powerful parasympathetico-mimetic drug with different chemical composition.

These drugs act directly on the effector cells and are not dependent on an intact nerve supply. When either is given, there is naturally a generalized response of all the end organs which are responsive to acetycholine. Clinically prominent among these are the sweat glands, salivary glands, bronchial mucous glands and musculature, increased peristalsis of the gastro-intestinal tract, and contraction of the vesical detrusor. There is, however, a variation in the intensity of reaction produced by different effector organs.[5]

These preparations are potent and alarming reactions can occur. They must not be given intravenously. The reaction on smooth muscle and glands can be terminated by intravenous injection or subcutaneous injection of .6 mg. atropine. The antagonistic action of atropine is more rapid with Mecholyl than with Doryl or Furmethide. In none of the cases in this study (total thirty injections) was there serious reaction, and atropine was never used.

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Parasympathetico-mimetic drugs should be used with caution in patients susceptible to asthma, as attacks are frequently precipitated. It is probably unwise to use any of these preparations in cardiac cripples or hyperthyroidism, as varying degrees of heart block have been reported experimentally with large doses.

Technic

On each patient the effects of these drugs was determined by making serial cystometrograms at 15 minutes, 30 minutes, 60 minutes, and 90 minutes after subcutaneous injection of each drug. Each experiment was preceded by a control cystometrogram, and the interval between testing each drug was at least 24 hours. Patients with normal and neurogenic bladders were studied.

A No. 18 straight catheter or a small Foley catheter with a 5 c.c. bag was inserted and the bladder contents emptied. A control cystometrogram was made, using a water manometer of the type described by Munro. Two per cent boric acid was allowed to run into the bladder at a rate of approximately 50 c.c. per minute. The pressure was checked on the manometer with each 50 c.c. increment, except in certain cases where 25 c.c. increments gave a more understandable record. Points checked on each normal bladder were sensation, first desire to void (FDV), capacity, manometric pressure at capacity, and maximal voluntary pressure (MVP). These observations were impossible in patients with neurogenic bladders, and vague complaints of distention, or leaking around the catheter were used as an end point. In no case was a bladder distended with more than 500 c.c. fluid. After the control cystometrogram .25 mg. of Doryl was injected subcutaneously, and tracings made as described. On subsequent days 5 mg. Furmethide and 25 mg. Mecholyl were similarly tested. The dosage given of each is the amount recommended by the manufacturer.

In all cases the individual experiments were concluded within ten days, and the control cystometrograms were essentially identical for each patient.

While no study was made of the effects on other organs than the bladder, the severity of reaction, as manifested by perspiration, salivation, increased peristalsis, flushing of the skin, etc., were subjectively noted and compared.

Results

Table I contains the essential figures from all the experiments. The validity of previously described effects of parasympathetico-mimetic drugs on the vesical detrusor is apparent. There is reduced

bladder capacity and increased tone. When sensation is present, the first desire to void occurs with less fluid in bladder. The maximal activity of the drugs is manifested in the first 15 minutes after injection, with gradual subsidence to a cystometric curve typical for that particular bladder in 90 to 120 minutes.

Diagrams 1, 2, 3, 4, show superimposed cystometrograms demonstrating graphically effects of Doryl, Mecholyl, and Furmethide. Examples of a normal bladder, a reflex bladder, an autonomous bladder and an atonic bladder are given.

It is apparent and uniform that Mecholyl is decidedly inferior in its effect on the bladder to either of the other two drugs studied. Furthermore, when given parenterally, the reaction to Mecholyl is relatively most unpleasant.

While not uniform, Doryl and Furmethide show approximately equal reaction on the detrusor. In four instances Doryl gave a more intense reaction, and in four Furmethide showed the stronger effect. The remaining two cases had almost identical figures. In no case were the variations great, and may have been due to absorption differences. (In all cases Furmethide was more distressing to the patient.)

Comment

Our objective has not been to present an argument for or against the use of parasympathetico-mimetic drugs, but only to present comparative data on three available drugs. It is regrettable that Urecholine was not included. The authors were unfamiliar with the latter drug when the study was commenced, and as yet have had no occasion for its use.

All three preparations may be given orally. Whether or not the same ratio or effectiveness is present with oral administration is unknown to use. Except for sporadic injections for acute urinary retention postoperatively in young people, our policy is to give these drugs orally. Our preference is Doryl tablets of 2 mg. size. This drug has been viewed with skepticism by some because of its pharmacologically demonstrable nicotinic action; i.e., stimulation of autonomic ganglia and striated muscle. We have never observed any such detrimental action in clinical use.

Whether tolerance to the action of these parasympathetico-mimetic drugs is developed after prolonged use is uncertain.

We have one patient, a 24 year old white school teacher, who has had a known atonic bladder for four years, and who formerly underwent daily catheterizations. Her residual urine was over

1,000 c.c. This was reduced to 150 to 200 c.c. twenty minutes after administration of 2 mg. Doryl by mouth. She took from 4 to 6 Doryl tablets daily for over 8 months without untoward effect. At the end of this time, no change could be detected cystoscopically or cystometrically. Her residual urine after Doryl had increased to 200 to 400 c.c. At no time was there urinary tract infection and her pyelograms were normal. We are unable to say whether tolerance to the drug was developing or the bladder atonicity was progressing.

SUMMARY

The effect on the bladder of parenterally given Doryl, Mecholyl, and Furmethide were compared using ten subjects, three of whom had normal bladders, and the remainder various types of neurogenic bladders.

Uniformly, Mecholyl showed a weaker response on the vesical detrusor than either Doryl or Furmethide. Of the latter two drugs, it was not possible to draw valid conclusions as to which had the stronger action. Both cause greatly increased tone, as manifested by higher intravesical pressure, lessened capacity, and a shift to the left of the first desire to void.

Unpleasant side reactions were most severe after injection of Mecholyl and least severe after injection of Doryl.

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SURGICAL MANAGEMENT OF URETEROPELVIC OBSTRUCTION*

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HYDRONEPHROSIS is a relatively common condition. In 330 male patients between the ages of 18 and 36, as reported by Deming,[1] it represented 8.8 per cent of renal lesions. In a study by H. A. Newman,[8] 23 of 85 soldiers with congenital anomalies of the urinary tract had hydronephrosis, believed to be due to ureteropelvic obstruction. Harrison and Botsford[6] found 21 cases of hydronephrosis among 72 examples of genito-urinary anomalies admitted to an Army Hospital. It is, therefore, important to be alert to the possibility of retention of urine in the renal pelvis in patients with symptoms suggestive of renal diseases. Too frequently the problem is dismissed because of a negative plain x-ray film of the abdomen. It is well to review the necessity of thorough investigation of the urinary tract when there is a suggestion of renal pathology, particularly in the younger age group.

SYMPTOMS

There are no characteristic symptoms of hydronephrosis due to ureteropelvic obstruction. The presenting complaints may be from obstruction or infection, and these patients are frequently perplexing diagnostic problems. In a series of 52 cases reported by Henline and Hawes,[12] 20 patients complained primarily of indigestion, flatulence or nausea. Pain in the back, varying in severity from a dull ache to renal colic, was present in 46 cases. Urinary symptoms (dysuria, hematuria, and increased frequency of urination) were present in only 22 cases, and in 19 of these 52 patients the urinalysis was completely normal.

The symptoms may be referred to the epigastrium or lower abdomen when there is a large hydronephrotic sac. Frequently, even a very large hydronephrosis cannot be palpated; when there is a palpable mass, it must be differentiated from other disease processes causing enlargement of the kidney. Ureteropelvic obstruction is usually diagnosed after an examination is performed to determine the cause of renal pain or tumor, explain hematuria or pyuria, or because vague gastro-intestinal symptoms are present.

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Pathology

The structural damage to the kidney varies from an insignificant pyelectasis to a pyonephrotic mass, depending entirely on the degree and duration of obstruction, and the severity of the infection. There are several types of mechanical obstruction which may be encountered. Stenosis of the ureteropelvic junction, either congenital or acquired, is generally conceded to be the most frequent. High implantation of the ureter into the renal pelvis is another cause of obstruction, but most writers attribute such faulty implantation to a primary ureteropelvic obstruction followed by dilatation and sagging of the renal pelvis. Other less frequent etiologic factors are hyperplasia of the ureteral muscle at the junction with the pelvis, fibrous bands across the ureter, accessory renal vessels, and congenital valves of the ureter. All of these lesions may exist singularly or in combination.

Surgical Management

It is only by complete relief of urinary stasis that satisfactory results may be obtained from plastic surgery on the renal pelvis. Primary obstruction may be complicated by secondary obstructive lesions which interfere with adequate drainage after the primary obstruction has been relieved. Examples of such secondary lesions are diverticula of the bladder, the tortuous, dilated ureter resulting from lower ureteral obstruction, or fibrous bands in the proximity of a stenotic ureteropelvic junction. In some cases it is difficult to determine which process is primary and which is secondary, but this must be determined before definitive surgery is undertaken.

Obstructive lesions in the ureteropelvic area are more easily corrected if the renal function has not been too greatly impaired before the hydronephrosis is recognized. A greatly dilated kidney with good function and one which is relatively free from infection may respond well to plastic procedure,[2] while a much smaller kidney, accompanied by infection and renal atrophy, will often progress to complete destruction, even after the obstruction is relieved. The choice between conservative surgery and nephrectomy depends chiefly on the following conditions: (1) The functional value of the obstructed kidney, (2) The condition of the opposite kidney, (3) The presence and severity of infection, (4) The age of the patient.

The disposition of a kidney severely damaged by hydronephrosis is a difficult problem. An accurate estimation of the potential function of this kidney after reconstruction cannot be made, and therefore, the condition of the opposite kidney must be a deciding factor in determining the type of treatment. The degree of compensatory

hypertrophy, if such has occurred, is a help in evaluating the duration of the disease and the extent of permanent renal damage. After an episode of obstruction or infection, the function of a diseased kidney may be temporarily reduced, but if there is no evidence of readjustment in the opposite kidney a conservative operation is usually indicated.

Infection is an important factor, but a superficial infection, in a patient who has an acid urine, is insignificant. When, however, the pelvis and ureter have become thickened from an infectious process which has permeated the renal parenchyma, little improvement in function can be expected. It is better to be more conservative in the younger patients, since the kidneys have a better recuperative power, and there is a greater need for conservation of renal tissue.

One of our outstanding failures resulted from operating on a hydronephrotic kidney in the presence of extensive infection. The patient was first seen by a general surgeon for acute abdominal pain and distention. At operation a large retroperitoneal cystic mass was found. The surgeon drained this cyst, and brought out a tube through a stab wound to the left of the midline incision. The patient was first seen by us a month later. The left hydronephrotic kidney was draining urine through the tube, and it was found to have fair function. At operation the peritoneal tissue was edematous, but the kidney was easily liberated. The wall of the very large renal pelvis was thickened and tore easily. The cortex was very thin. A plastic operation was performed, the redundant pelvis excised and the ureter reimplanted into the pelvis. The convalescence was stormy and prolonged. Two months later, when the patient returned because of fever and a persistent fistula, the newly formed ureteropelvic outlet had become completely occluded, and the kidney was a pyonephrotic mass. We had apparently placed too much reliance on the antibiotics, and on the patient's desire for a quick recovery. If, at the first operation, when the condition of the kidney was recognized, nephrostomy drainage had been provided, it is probable that within two or three months the kidney would have recovered to the extent that a successful operation could have been done.

A second patient, a 25 year old man who entered the hospital because of pain in his right lumbar area had a history of repeated attacks of pyelitis. At the age of seven his left kidney had been removed because of hydronephrosis. The function of this right kidney was good at that time, but no pyelogram was made. When he came under our care he had a minute calculus in the lower calix of his remaining kidney, and the renal pelvis was considerably dilated. At operation the stone could not be located, and since the patient

had only one kidney an extensive search was considered unwise. Some adhesions were liberated, and the constriction at the uteropelvic junction was divided longitudinally and sutured transversely. Nephrostomy drainage was instituted, but renal drainage, while improved, was never satisfactory, and the infection persisted. The stone grew rapidly, and twelve months later another operation was advised. This time the stone was removed, the adhesions again liberated, the ureter splinted, and a nephropexy done. Now, three years following the last operation, the patient feels well, and has only a few pus cells in his urine. Renal function and drainage are satisfactory. The ureter was large and redundant, and it is probable that nephropexy and a ureteral splint at the first operation would have given a better result.

When there is obvious or suspected bilateral disease, a nephrectomy should not be performed unless one kidney is entirely destroyed. In doubtful cases a nephrostomy on the poorer kidney is advisable, while the function of the other kidney is being restored by plastic operations. The bad kidney may then be removed or treated conservatively depending on its functional value and the degree of success in treating the good kidney.

Surgical correction of potential hydronephrosis is not indicated. Patients having slightly dilated renal pelves, with pyelographic evidence of narrowing or distortion of the pelvic orifice, should be kept under observation unless there is persistent pain or recurrent infection. Frequently, and especially in adults, the condition does not progress and the renal function remains normal. Plastic efforts in such cases often fail to improve either appearance or function.

Operative Technic

Dr. Frank Hinman[6] notes that more than 25 surgeons have proposed a method of ureteropyeloplasty or some modification thereof. This not only indicates interest in the problem, but testifies to the multiplicity of problems that may be encountered.

There is considerable difference of opinion regarding splinting of the ureter and nephrostomy drainage. The following quotations are examples: "Uninfected hydronephrotic kidneys should not be insulted at the time of plastic procedures by producing infection with the use of drainage tubes and splints" (Deming).[2] "No matter how generous the surgeon is in the use of suture and plastic repairs, the outcome is likely to be a failure unless he makes use of ureteral splinting by catheter; conversely, if hep laces his dependence in splinting the results are almost certain to be successful, even though he uses no sutures at all" (Gibson).[4] Gibson also states

that statistical reviews indicate failure in 13 to 33 per cent of the cases. It is doubtful whether the use of splints or the lack of splints is responsible for this high degree of failure. Failure results from improper selection of cases, and lack of reconstruction of the ureteropelvic junction in such manner that dependent drainage is accomplished.

Elaborate plastic procedures are rarely necessary. There are really only two main types of intrinsic ureteropelvic obstructions: stricture of the uteropelvic junction, and valve formation caused by high insertion of the ureter into the renal pelvis. Adhesions, either congenital or acquired, which involve the renal pelvis and upper ureter may cause obstructive kinks at or near the pelvic orifice with retention of urine and dilatation of the kidney. Not infrequently such obstruction is complicated by a low position or mobility of the kidney. The liberation of such adhesions, completely freeing the upper ureter and pelvis, with the addition of careful nephropexy, will correct the faulty drainage unless sufficient dilatation of the renal pelvis has occurred to cause the ureter to emerge from the pelvis above the most dependent portion, or unless there are other obstructive lesions (Case One). Aberrant vessels to the lower pole of the kidney may play only an accessory role in the production of renal obstruction, serving as a band over which the ureter may be kinked in cases of nephroptosis, or as a secondary obstruction to an already enlarged renal pelvis. Only rarely are they the sole cause of obstruction.

Some controversy has arisen regarding the disposition of aberrant vessels. There are those who believe that all polar vessels should be preserved by plastic operations on the renal pelvis designed to remove the ureteropelvic area from contact with the blood vessels. This consists of division of the ureter or pelvis and reanastomosis in front of the blood vessel. There are others who believe that in most cases aberrant vessels producing obstruction should be divided and ligated. Neither view should be strictly adhered to. If the artery is small it may be divided without noticeable change to the kidney. Compression of the vessel and observation of the size of the renal area which changes color gives one an idea of the amount of renal tissue which might become ischemic. Should an area equal to one-fifth of the renal surface change color the artery should be preserved. In the presence of pronounced infection there is a greater danger in dividing an aberrant artery. In the case of large vessels which cannot be disposed of except by ligation, reimplantation of the ureter in front of the artery is the best procedure (Case Two). It must be remembered that stenosis of the ureteropelvic junction may accompany either adhesions or aberrant vessels,

and appropriate plastic procedures are indicated where this is found.

When the stricture of the ureter or the ureteropelvic junction, or when the renal pelvis has become so dilated that the ureter emerges from an area above the most dependent part of the renal pelvis, there are several operations to enlarge the pelvic outlet and secure dependent drainage. The ureter may be divided below the stricture and reimplanted at the most dependent portion of the renal pelvis; the stricture may be incised longitudinally and sutured transversely, or one may select one of the more elaborate procedures such as recommended by Schwyzer or Foley.[3] In the hands of most of us, the simpler the procedure the better the results. D. M. Davis, T. E. Gibson, and Robert McIver have demonstrated that excellent results can be obtained by merely dividing the stricture and splinting the area without the use of sutures. This method is particularly recommended when the tissues are greatly thickened, when there is acute infection, or when the area of constriction is unusually long. When the ureter emerges high on the pelvic wall, we have elected to remove an elliptical portion of the pelvis below the ureter. When the margins are approximated, the pelvic outlet is brought to a more dependent level and the constriction can be dealt with by simple division and suturing in the opposite direction, or by division and splinting.

Whenever the hydronephrosis is so large that the redundancy of the pelvic wall may interfere with drainage, partial resection of the pelvic wall should be carried out (Case Three). Sections may be excised from the anterior and posterior walls in such a manner that the ureter will emerge from the most dependent area. The entire pelvis and ureter may be excised leaving just enough margin to make an accurate closure without tension. The ureter is then reimplanted in the most dependent area of the reconstructed pelvis.

In plastic operations on the renal pelvis and ureter, very fine sutures such as No. 0000 catgut on an atraumatic needle should be used. Sutures should be placed accurately and tightly enough to maintain surface contact. They should not pierce the mucous membrane. In most cases we recommend the use of a nephrostomy tube and a splinting ureteral catheter. The proper splinting catheter is a number 10 or 12 French red rubber catheter which should extend hown the ureter about 4 to 6 inches. A window should be cut where the catheter traverses the renal pelvis. A small Malecot or Foley catheter should be used for a nephrostomy tube. Both catheters should be brought out through the renal parenchyma in the lower pole of the kidney. In all cases the operation is completed by a nephropexy and we prefer the method as described by Deming. The

kidney is placed sufficiently high to prevent redundancy of the ureter, and an effort is made to fix the lower pole of the kidney as far laterally as possible.

Drainage and splinting should be maintained from two to six weeks, depending on the severity of the infection. Usually the splinting catheter is removed first and the nephrostomy tube retained until normal emptying of the renal pelvis has been demonstrated.

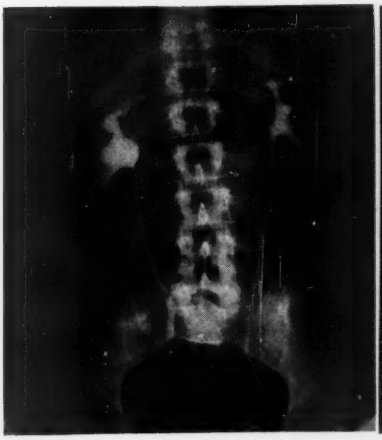


Fig. 1. Preoperative retrograde pyelogram (Case 1).

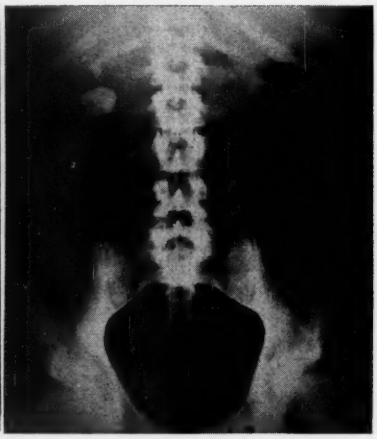


Fig. 2. Preoperative excretory urogram (Case 1).

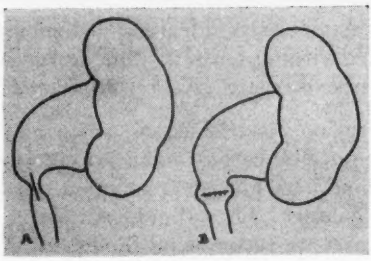


Fig. 3. Illustration of findings and operation (Case 1). (From Dodson, A. I.: Urological Surgery. St. Louis: The C. V. Mosby Co., 1949.)

Illustrative Cases

Case 1. R. S., No. B59057. Intermittent dull aching pain in the right flank of 3 years' duration. Figures 1 and 2 show preoperative retrograde and excretory urograms.

At operation (fig. 3) the right kidney and upper ureter were carefully ex-

posed and cleaned. The pelvis was moderately dilated, and there was narrowing at the ureteropelvic junction. A longitudinal incision one centimeter long

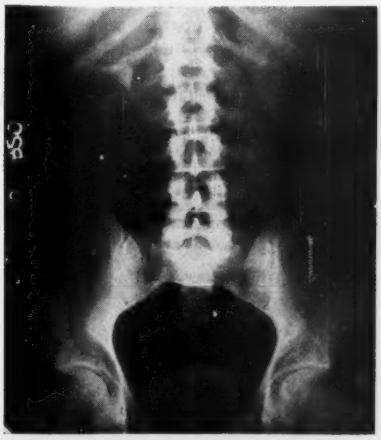


Fig. 4. One year postoperative excretory urogram (Case 1).

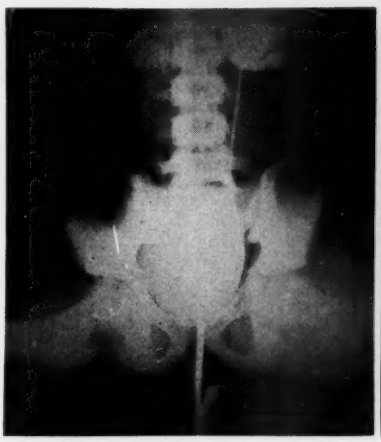


Fig. 5. Preoperative retrograde pyelogram (Case 2).

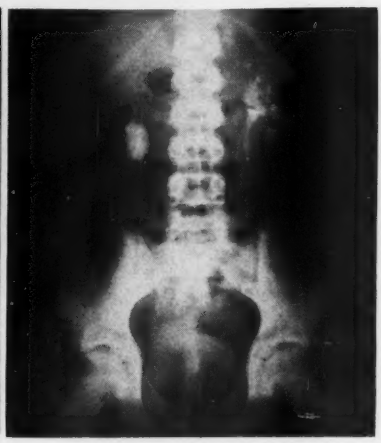


Fig. 6. Four months postoperative film from Case 2. Note the presence of minimal ureteropelvic obstruction on the right side.

was made in the ureteropelvic area and the wound sutured transversely. A nephrostomy tube and a ureteral splint were inserted. Nephroplexy was performed by the Deming method.

Splinting ureteral catheter was left in place for 18 days.

Figure 4 is a one year postoperative excretory urogram, 20 minute film.

CASE 2. R. A., No. 71821. Pain in the left flank region, with nausea and vomiting.

Figure 5 is a preoperative retrograde pyelogram.

At operation the kidney and upper ureter were cleaned of fat and fibrous tissue. A large aberrant vessel to the lower pole was found. The redundant pelvis was resected and the ureter reimplanted in front of the vessel. This did not completely relieve the obstruction. Ligation of a vessel of this size was impractical. The obstruction was relieved by flexing the kidney parenchyma posteriorly and anchoring the upper pole to the lower pole by a single suture through the renal capsule. Nephropexy was then performed.

A splinting catheter was left in the reimplanted ureter for 14 days.

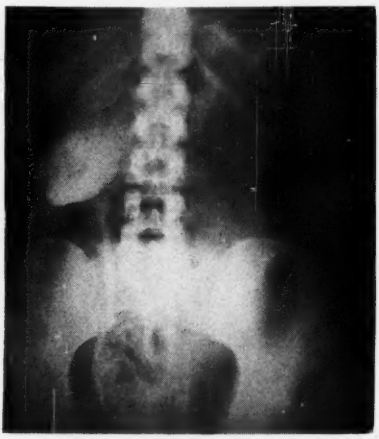


Fig. 7. Preoperative retrograde pyelogram (Case 3).

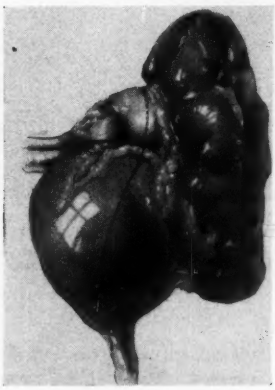


Fig. 8. Line of resection of renal pelvis (Case 3).

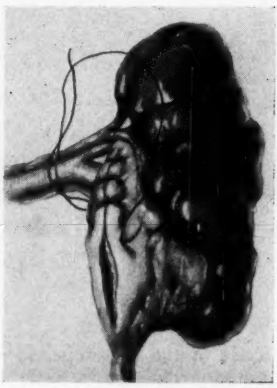


Fig. 9. Suturing of renal pelvis (Case 3). (From Dodson, A. I.: Urological Surgery. St. Louis: The C. V. Mosby Co., 1949.)

Figure 6 is a four months postoperative film.

CASE 3. G. F., No. B31449, 22 year old female who complained of pain in right flank and pyuria of three years' duration.

Figure 7 is a preoperative retrograde pyelogram.

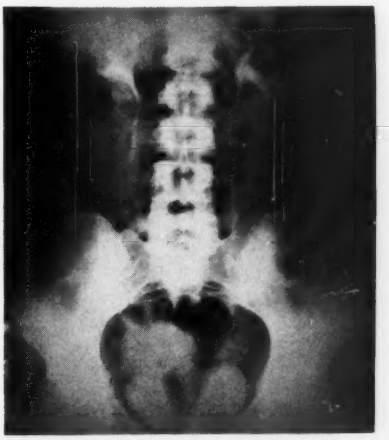


Fig. 10. Postoperative film (Case 3).

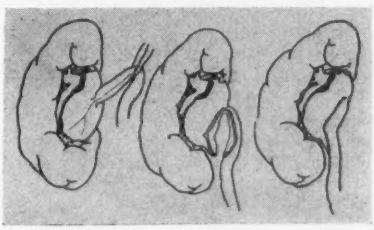


Fig. 11. Diagrammatic repair of ureteropelvic by the Foley method. (From Dodson, A. I.: Urological Surgery. St. Louis: The C. V. Mosby Co., 1949.)

At operation (figs. 8 and 9), after cleaning the upper ureter, pelvis and kidney, no obstructive lesion was found. The redundant pelvis was resected without disturbing the ureteropelvic junction. A nephrostomy tube was inserted and the pelvis closed. A careful nephropexy by the Deming technic was performed. The nephrostomy tube was removed on the fourteenth postoperative day.

Figure 10 reveals the postoperative results.

CONCLUSIONS

Hydronephrosis due to mechanical obstruction at the ureteropelvic junction is a fairly frequent and a serious genito-urinary pathologic entity. The etiology is one, or a combination of several fac-

TABLE 1

Essential figures from all the tests. First desire to void, bladder capacity, manometric pressure at capacity in centimeters of water, and maximal voluntary pressure, at 15 minutes, 30 minutes and one hour after injection of each drug.

		15 minutes after injection				30 minutes after injection				1 hour after injection				
		First Desire to Void	Capacity	Pressure in cm. H_2O at Capacity	Maximum Voluntary Pressure	First Desire to Void	Capacity	Pressure in cm. H_2O at Capacity	Maximum Voluntary Pressure	First Desire to Void	Capacity	Pressure in cm. H_2O at Capacity	Maximum Voluntary Pressure	Reaction
Normal Bladder	CONTROL	200	300	9	50+									
	Doryl	75	125	24	50+	150	300	22	50+	150	300	15	50+	Slight
H. R., age 52	Furmethide	50	100	38	50+	100	200	25	50+	150	300	14	50+	Moderate
Female	Mecholyl	175	300	9	50+	200	300	10	50+	200	300	8	50+	Severe
Normal Bladder	CONTROL	250	300	8	50+									
	Doryl	75	100	12	50+	100	125	12	50+	100	250	10	50+	Slight
C. O., age 64	Furmethide	25	50	20	50+	50	100	8	50+	125	175	10	50+	Moderate
Male	Mecholyl	100	150	10	50+	100	150	8	50+	150	250	10	50+	Severe
Normal Bladder	CONTROL	150	350	9	50+									
	Doryl	75	175	15	50+	200	350	17	50+	200	350	15	50+	Slight
W. J., age 17	Furmethide	50	100	25	50+	75	100	18	50+	100	275	15	50+	Moderate
Male	Mecholyl	200	325	14	50+	200	350	11	50+	200	350	10	50+	Severe
Autonomous Bladder	CONTROL	No	450	42	Leaked									
	Doryl	No	50	40	Leaked	No	150	36	Leaked	No	300	42	Leaked	Slight
H. L., age 36	Furmethide	No	75	50	Leaked	No	150	38	Leaked	No	350	50	Leaked	Moderate
Male	Mecholyl	No	150	42	Leaked	No	350	38	Leaked	No	450	40	Leaked	Severe

TABLE 1 (Continued)

Autonomous Bladder	CONTROL	No	350	30	Leaked								
	Doryl	No	25	40	Leaked	No	75	40	Leaked	No	175	40	Leaked	Slight
T. B., age 42	Furmethide	No	75	50	Leaked	No	25	40	Leaked	No	75	40	Leaked	Moderate
Male	Mecholyl	No	150	22	Leaked	No	175	18	Leaked	No	350	32	Leaked	Severe
Reflex Bladder	CONTROL	No	150	4										
	Doryl	No	75	10		No	75	22		No	100	12		Slight
B. C., age 38	Furmethide	No	35	10		No	75	24		No	100	10		Moderate
Male	Mecholyl	No	150	9		No	150	6		No	150	7		Severe
Hypotonic Bladder	CONTROL	No	500	22	50+									
	Doryl	No	200	44	50+	No	350	16	50+	No	400	14	50+	Slight
R. A., age 36	Furmethide	No	300	30	50+	No	350	28	50+	No	350	27	50+	Moderate
Male	Mecholyl	No	250	33	50+	No	400	16	50+	No	400	14	50+	Severe
Autonomous Bladder	CONTROL	No	500	27	50+									
	Doryl	No	100	44	50+	No	75	38	50+	No	500	29	50+	Slight
M. R., age 34	Furmethide	No	150	38	50+	No	350	28	50+	No	500	28	50+	Moderate
Female	Mecholyl	No	250	26	50+	No	400	24	50+	No	500	27	50+	Severe
Atonic Bladder	CONTROL	No	500+	10	20									
	Doryl	No	150	40	Leaked	No	200	40	Leaked	No	500+	16	20	Slight
D. T., age 22	Furmethide	No	500	30	34	No	500	23	30	No	500+	10	20	Moderate
Female	Mecholyl	No	500+	16	20	No	500+	14	20	No	500+	10	20	Severe
Hypotonic Bladder	CONTROL	350	500	10	25									
	Doryl	100	200	43	48	350	500	22	28	350	500	22	28	Slight
J. D., age 24	Furmethide	100	200	40	46	300	350	35	39	300	500	18	26	Moderate
Male	Mecholyl	350	500	19	24	350	500	12	19	350	500	10	18	Severe

DIAGRAM 1

Diagram 1. Normal Bladder. Control cystometrogram and composite cystometrograms at 15 minutes, 30 minutes, and one hour after injection of Doryl, Furmethide and Mecholyl.

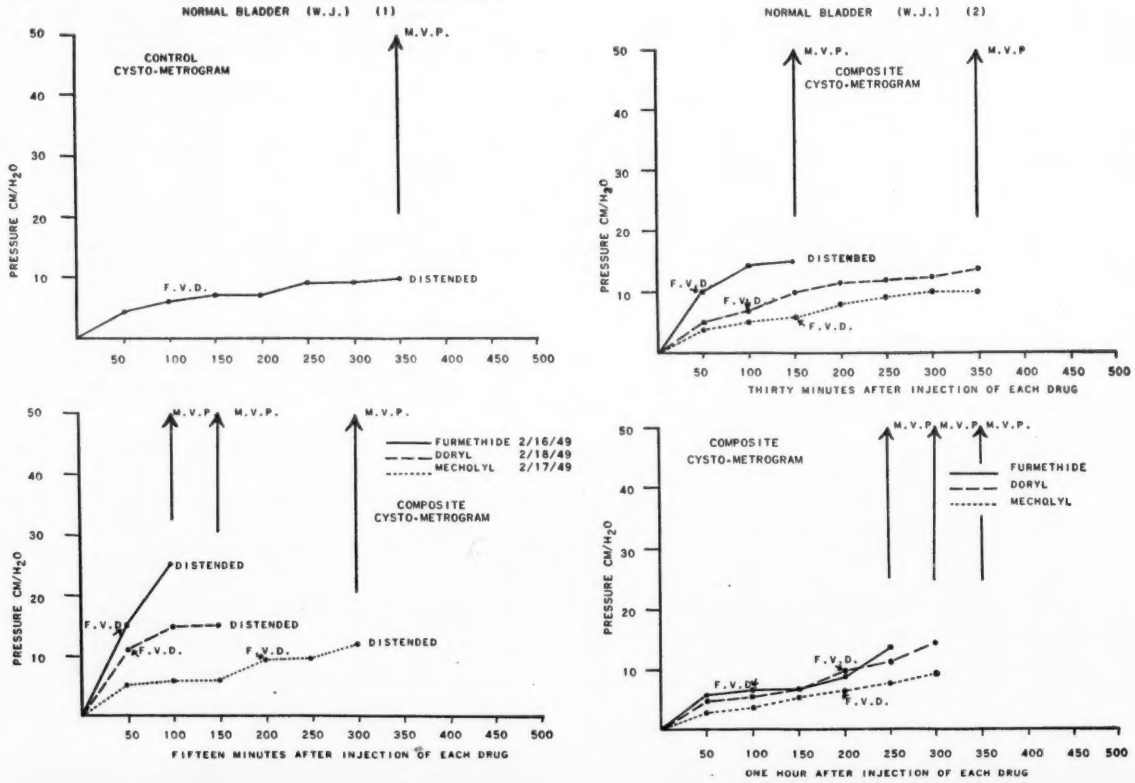


DIAGRAM 2

Diagram 2. Reflex neurogenic bladder. Control cystometrogram and composite cystometrograms 15 minutes, 30 minutes and one hour after injection of each drug.

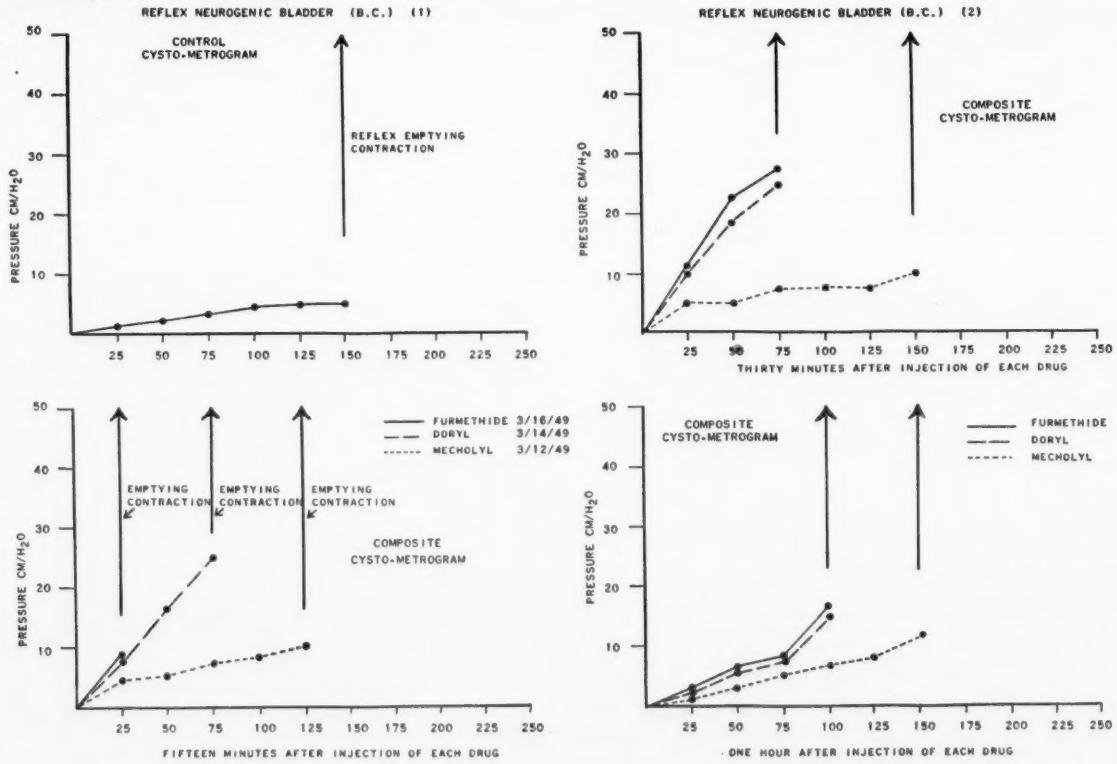


DIAGRAM 3

Diagram 3. Atonic neurogenic bladder. Control cystometrogram, followed by composite cystometrograms 15 minutes, 30 minutes and one hour after injection of each drug.

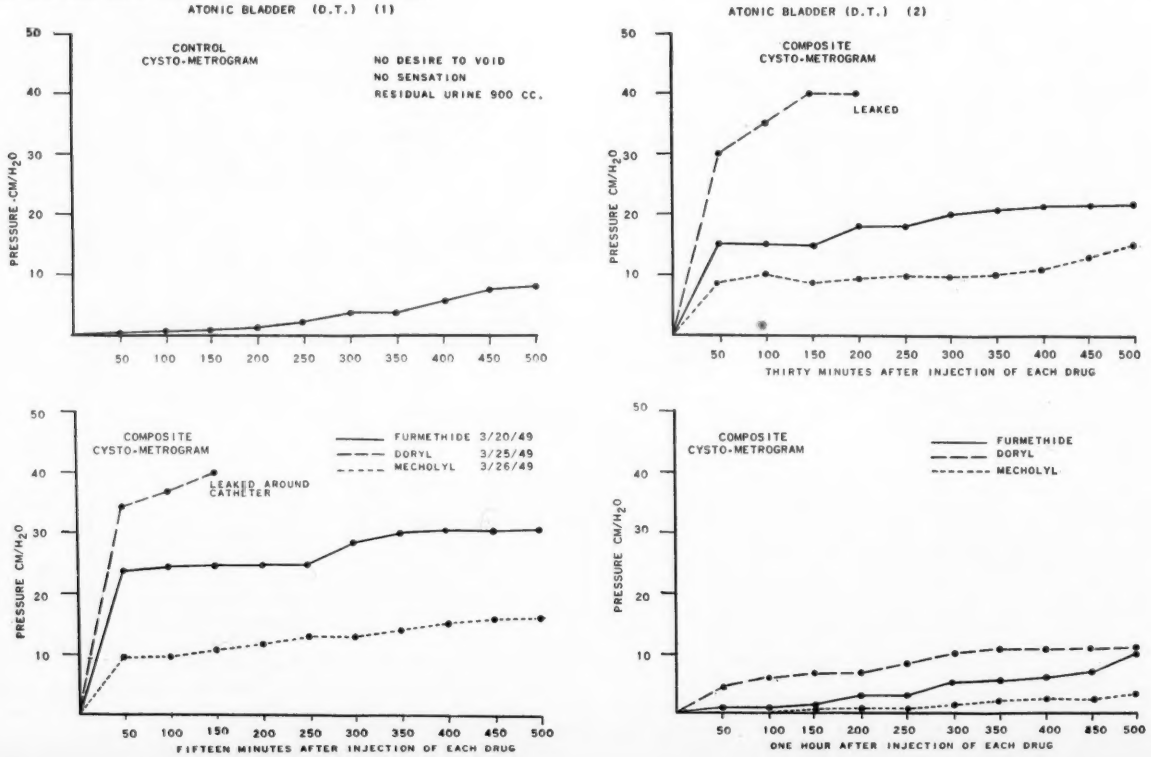
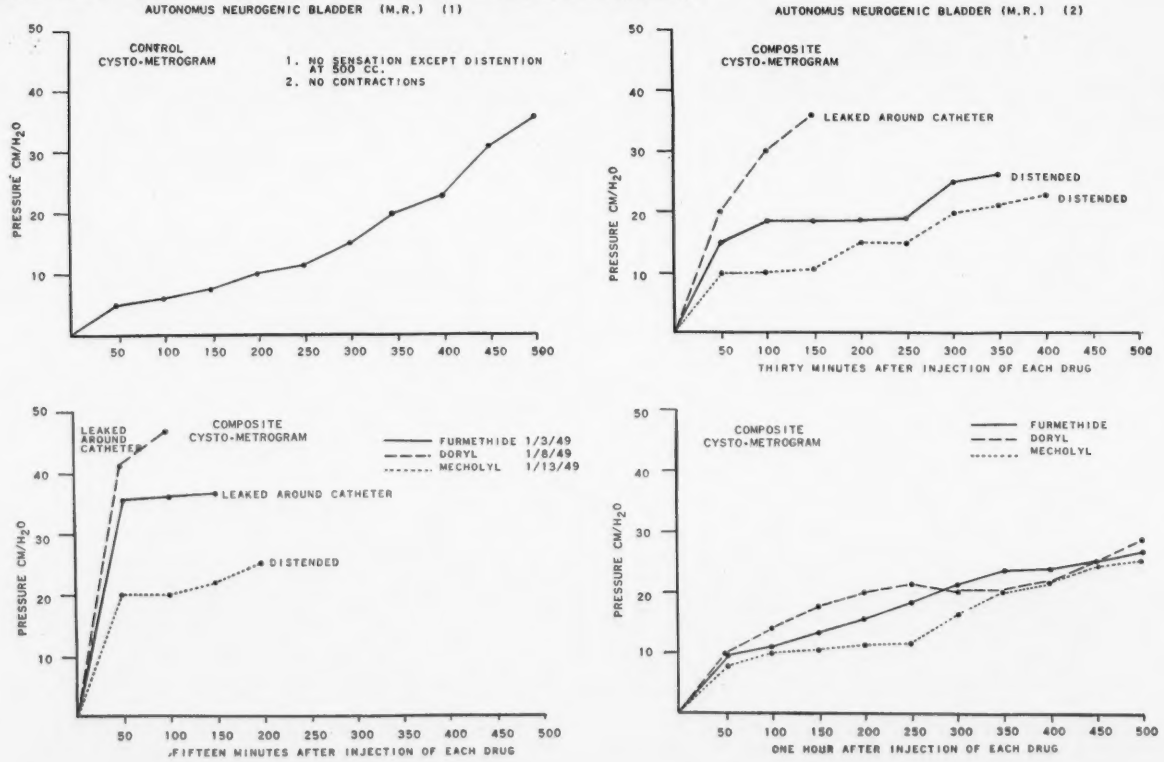


DIAGRAM 4

Diagram 4. Autonomous neurogenic bladder. Control cystometrogram, followed by composite cystometrograms 15 minutes, 30 minutes and one hour after injection of Doryl, Furmethide, and Mecholyl.



tors, most frequent of which are: stenosis of the ureter at its junction with the pelvis, high implantation of the ureter, fibrous bands in the ureteropelvic area, aberrant blood vessels to the lower pole of the kidney, and congenital valves of the ureter. There is a high incidence of involvement of both kidneys with this condition.

The diagnosis depends on urography. The type of obstruction can usually be determined from retrograde pyelograms and delayed emptying films, but final judgment is withheld until operation.

Careful evaluation of the degree of renal damage and the potential function of an obstructed kidney is essential if satisfactory results are to be obtained. The duration of obstruction, the severity and type of infection, the presence or absence of compensatory hypertrophy of the opposite kidney, and the recovery of function on ureteral catheter drainage or nephrostomy are factors to be considered in estimating the value of a damaged kidney. Conservative surgery is preferable in the younger age group because of a greater restorative power of the renal parenchyma and the high incidence of these lesions being or becoming bilateral.

For successful results, complete elimination of urinary stasis is essential, regardless of the type of operation elected.

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THE MEDICAL COLLEGE OF VIRGINIA NUMBER

THE SOUTHERN SURGEON has highly honored the School of Medicine of the Medical College of Virginia in offering it the privilege of sponsoring this number of the journal devoted to the publication of articles prepared by members of its faculty. We are deeply appreciative of this gracious and generous act of friendliness and sincerely hope that the presentations in this "Medical College of Virginia Number" will not only be of interest and benefit to its readers but also in keeping with the high standards which have consistently characterized contributions appearing in THE SOUTHERN SURGEON.

We feel that we have much in common with this distinguished journal. From its very beginning members of our faculty have been privileged to have their manuscripts accepted by it, to serve on its Editorial Board, and by other means to further the advancement of surgery, particularly in the South. Like THE SOUTHERN SURGEON we of this medical community also aspire to encourage younger members of the surgical profession by making available to them adequate clinical, laboratory and research facilities in a friendly, stimulating atmosphere.

The College has been blest through the years in having as mem-

bers of its Faculty so many outstanding leaders in surgery. It is with pride that the writer recalls in his own time as a student and alumnus, the names of a host of such illustrious physicians who were in the vanguard of those dedicated to the advancement of surgery and who have played such a major role in making Richmond one of the great medical centers of the South. To our great sorrow and loss many of this group have now passed away. Among these were such distinguished surgeons and revered teachers as Stuart McGuire, George Ben Johnston, A. Murat Willis, Paul LaRoque, J. Shelton Horsley and W. Lowndes Peple. Today in Richmond, in the Southland and throughout the nation, their followers, many of whom were their contemporaries and former pupils, are ably and with great vision and appropriate sense of responsibility extending, in the best traditional fashion, the art and science of surgery.

In light of all this it is understandable why we are particularly proud and grateful for this opportunity of sponsoring this issue of THE SOUTHERN SURGEON with a series of contributions bearing on surgery by members of our faculty.

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